

# Pain-related Insomnia Versus Primary Insomnia

## *A Comparison Study of Sleep Pattern, Psychological Characteristics, and Cognitive-behavioral Processes*

Nicole K. Y. Tang, DPhil,\*† Claire E. Goodchild, PhD,† Joan Hester, MBBS, FRCA, MSc,‡ and Paul M. Salkovskis, PhD†,§

**Background:** Recent applications of cognitive-behavior therapy for primary insomnia in the management of pain-related insomnia are based on the implicit assumption that the 2 types of insomnia share the same presentation and maintaining mechanisms. The objectives of this study were to compare the characteristics of patients who have pain-related insomnia with those reporting primary insomnia and to identify psychological factors that predict pain-related insomnia.

**Methods:** Chronic pain patients with concomitant insomnia (n = 137; Pain-related Insomnia Group) completed a selection of questionnaires that measure sleep patterns, psychological attributes, and cognitive-behavioral processes associated with the persistence of insomnia. Their responses were compared with those of primary insomnia patients (n = 33; Primary Insomnia Group), using 3 sets of multivariate analyses of covariance that took account of demographic differences. Hierarchical regression analyses were performed to identify predictors of insomnia severity among the chronic pain patients.

**Results:** The Pain-related Insomnia Group did not differ from the Primary Insomnia Group in their pattern and severity of sleep disturbance. The 2 groups were largely comparable in terms of their psychological characteristics, except that the Primary Insomnia Group was distinguishable from the Pain-related Insomnia Group by their greater tendency to worry. Patients in the Pain-related Insomnia Group reported levels of sleep-related anxiety and presleep somatic arousal that matched with those reported by patients in the Primary Insomnia Group. However, relative to patients in the Pain-related Insomnia Group, those in the Primary Insomnia Group reported more dysfunctional sleep beliefs and presleep cognitive arousal. In addition to pain intensity, depression, and presleep cognitive arousal were significant predictors of insomnia severity within the Pain-related Insomnia Group.

**Conclusions:** There are more similarities than differences between the 2 types of insomnia. Besides pain, mood, and presleep, thought processes also seem to have a role in the manifestation of pain-related insomnia. It is suggested that hybrid treatments that seek to

simultaneously address factors across these domains may represent more effective treatments than 1-dimensional interventions.

**Key Words:** pain, chronic pain, insomnia, cognitive-behavior therapy for primary insomnia, depression, health anxiety, presleep arousal, worry

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For many years, insomnia that develops after the onset of pain was known as “secondary insomnia.” Treatments for pain patients with concomitant insomnia tended to focus on pain reduction because insomnia was believed to be a symptom that would fade when pain was eliminated. This strategy is problematic as, first, chronic pain by definition is intractable, and second, many patients continue to experience difficulty sleeping even when good pain control is achieved.<sup>1–3</sup> Increasingly, improved sleep is seen by patients as an important treatment outcome.<sup>4,5</sup> There is an indisputable need to reconsider the conventional approach to pain-related insomnia.

The term “comorbid insomnia” has recently been recommended due to concerns that the use of “secondary insomnia” may promote undertreatment.<sup>6</sup> This shift in thinking has resulted in more proactive attempts to address sleep problems in chronic pain patients, including the application of cognitive-behavior therapy for primary insomnia (CBT-I) to pain-related insomnia.<sup>7</sup> CBT-I is a multicomponent treatment. It typically involves the use of empirically evaluated behavioral interventions (eg, stimulus control therapy and sleep restriction therapy) to help the patient drop unhelpful sleep practices, reassociate the bed/bedroom with sleep, consolidate fragmented sleep, and establish a new sleep-wake schedule. The cognitive component of the intervention addresses sleep-related anxiety. This can be achieved by helping the patient alter unhelpful sleep beliefs and manage unwanted presleep tension and worries. The notion of CBT-I is to reverse or eliminate the cognitive-behavioral factors perpetuating insomnia.<sup>8–14</sup> The effect of the treatment is robust and there is ample evidence supporting CBT-I as the choice of intervention for chronic insomnia.<sup>15–19</sup> The recent application of CBT-I to pain-related insomnia is pragmatic and is encouraged by the similar presentations between primary and pain-related insomnia.<sup>20–22</sup> Such development is also based on the assumptions that (1) patients with primary and pain-related insomnia shared similar psychological characteristics and that (2) the cognitive-behavioral mechanisms maintaining

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From the \*Arthritis Research UK Primary Care Centre, Primary Care Sciences, Keele University, Staffordshire; †Department of Psychology, Institute of Psychiatry, King's College London; ‡Pain Relief Unit, King's College Hospital, London; and §Department of Psychology, University of Bath, Bath, UK.

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Reprints: Nicole K. Y. Tang, DPhil, Arthritis Research UK Primary Care Centre, Primary Care Sciences Keele University, Staffordshire, ST5 5BG, UK (e-mail: n.k.y.tang@cphc.keele.ac.uk).

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primary insomnia also operate in pain-related insomnia. These assumptions, however, have not been tested.

Despite a surge in research on the reciprocal link between pain and sleep, factors characterizing and perpetuating pain-related insomnia have not been very well documented. Evidence showing similarities (ie, supporting the direct application of CBT-I to treat pain-related insomnia) and differences (ie, indicating a need for adaptation) between primary and pain-related insomnia is mostly scattered and indirect. A more consistent finding thus far is that poor-sleeping chronic pain patients report higher levels of pain<sup>23,24</sup> and manifest elevated levels of anxiety, depression, and preoccupation with health, compared to good-sleeping chronic pain patients.<sup>22,25,26</sup> It is not known whether these observed elevations in psychopathology match with those in primary insomnia and whether these general psychological characteristics help explain the severity of pain-related insomnia. Research has only just begun to investigate the specific processes perpetuating pain-related insomnia. A few cross-sectional studies have usefully demonstrated the presence of dysfunctional beliefs about sleep in fibromyalgia patients with problems sleeping,<sup>27</sup> and implicated a role for presleep arousal<sup>28,29</sup> and affective responses to pain and health<sup>22</sup> in pain-related insomnia. However, none of these studies made a direct comparison between primary and pain-related insomnia and thus potential differences between the 2 types of insomnia in these cognitive-behavioral processes remain to be identified.

To fill these gaps in the literature, the current study aimed to evaluate the similarities and differences between primary and pain-related insomnia directly. We compared patients with primary insomnia with chronic pain patients who have comorbid insomnia in terms of their (1) typical sleep pattern, (2) general psychological characteristics (eg, anxiety, depression, health anxiety, and tendency to worry), and (3) specific cognitive-behavioral processes linked to the perpetuation of insomnia (eg, sleep-related anxiety, dysfunctional beliefs about sleep, and presleep arousal). Based on findings of previous polysomnography-based and questionnaire-based studies,<sup>21,22,25,26,30</sup> we did not anticipate any significant differences in typical sleep pattern and general psychological characteristics between the Pain-related Insomnia Group and the Primary Insomnia Group. We, however, predicted that the 2 groups would differ on some of the cognitive-behavioral processes, as all of these constructs were developed based on research focusing on primary insomnia. In the absence of a definitive model of pain-related insomnia, exploratory hierarchical regression analyses were conducted to identify psychological predictors of pain-related insomnia (after the effect of pain was accounted for).

## METHODS

### Overview

Systematic comparisons were carried out on questionnaire data collected from (1) chronic pain patients with insomnia (referred to below as “Pain-related Insomnia Group”) and (2) patients with primary insomnia (referred to below as “Primary Insomnia Group”). Chronic pain patients with insomnia were recruited from a hospital-based pain clinic, whereas primary insomnia patients were recruited from 7 general practices located in the same

catchment area. The protocol of the study had full ethical approval.

### Participants

All participants completed and returned a questionnaire together with written informed consent to take part in the study. Inclusion criteria for the Pain-related Insomnia Group were: (1) 18 to 65 years of age (because our research focused on working-age adults); (2) literate in English; (3) nonmalignant pain of a duration of at least 6 months; and (4) scoring 15 or above on the Insomnia Severity Scale (ISI<sup>31</sup>). Patients were not invited to take part in the study if they reported (1) having received an injection or operation for their pain in the last month; (2) suffering from severe psychiatric/psychological disorders; or (3) visual/cognitive impairments that rendered completing the questionnaire unfeasible. The inclusion and exclusion criteria for the Primary Insomnia Group were identical, except that patients would have been excluded from the study had they reported any pain of 3 months or longer.

In addition to the ISI, all participants included in the study were administered the Duke structured interview schedule for DSM-IV-TR and ICSD-2 sleep disorder diagnoses<sup>32</sup> to establish the presence of clinical insomnia. The interview took approximately 1 to 1.5 hours to complete and was administered either in person or over the phone. The purpose of this additional procedure was to confirm that all insomnia problems met diagnostic criteria for duration (> 1 mo), frequency (> 3 nights/wk), and severity (causing clinically significant interference), and that the patients had no other medical (except chronic pain for the *Pain-related Insomnia Group*), psychiatric, or sleep disorders that could better account for their sleep disturbance.

The *Primary Insomnia Group* comprised 137 of the 515 patients approached at the pain clinic, representing an uptake rate of 27% (74 of the patients approached declined the invitation to take part, 271 patients did not return the questionnaire, 33 patients returned the questionnaire but did not meet criteria for clinical insomnia). The majority of these patients reported having more than 1 pain (86.9%). The most commonly cited area of pain was lower back (70.1%), followed by legs (55.5%), neck (36.5%), knees (33.6%), shoulders (32.1%), joints and arms (23.4%), upper back (22.6%), head (17.5%), hands (10.2%), hip (8.8%), feet (8%), and abdomen (7.3%). The *Primary Insomnia Group* comprised 31 of the 56 participants who returned a completed questionnaire, representing an uptake rate of 55% (20 were excluded because they reported chronic pain, 5 did not meet criteria for clinical insomnia). None of the patients in the *Primary Insomnia Group* reported any pain.

### Measures

The research questionnaire contained a selection of validated measures that assess sleep and psychological characteristics of the participants, as well as cognitive-behavioral processes linked to the maintenance of insomnia. Each of these measures is briefly described below.

#### Measures of Psychological Characteristics

Three questionnaires were used to index the participants' levels of anxiety, depression, health anxiety, and tendency to worry.

#### *Hospital Anxiety and Depression Scale (HADS<sup>33</sup>)*

The HADS contains 14 items describing anxiety and depression symptoms in nonpsychiatric medical contexts.

Participants were asked to rate the severity of their symptoms during the past week on a 4-point scale (0 to 3), generating a score for both “Anxiety” (range, 0 to 21) and “Depression” (range, 0 to 21). Higher scores indicate greater symptom severity. The HADS has demonstrated good internal consistency (mean Cronbach  $\alpha > 0.8$  for both subscales) and concurrent validity (agreement with clinician ratings of anxiety:  $r = 0.54$  and depression:  $r = 0.79$ ).

#### **Short Health Anxiety Inventory (SHA<sup>34</sup>)**

The SHAI comprises 14 groups of 4 statements concerning health anxiety (ranked 0 to 3). Participants were asked to select the statement most appropriate for them from each group. The rank scores were then summed to give a total score (range, 0 to 42), with a higher score indicative of a higher level of health anxiety. The SHAI has demonstrated good internal consistency (Cronbach  $\alpha = 0.89$ ).

#### **Penn State Worry Questionnaire (PSWQ<sup>35</sup>)**

The PSWQ contains 16 statements. Participants in the present study were asked to rate how typical each of the statements (eg, “Once I start worrying I cannot stop”) was of them using a 5-point scale, where 1 means “Not typical of me” and 5 means “Very typical of me.” The total score ranges from 16 to 80, with a higher score indicating a greater tendency to worry. The PSWQ has been demonstrated to be a highly reliable measure (Cronbach  $\alpha \geq 0.91$ ; test-retest reliability:  $r \geq 0.74$ ).

### **Measures of Sleep Patterns and Cognitive-behavioral Processes**

Four questionnaires were used to assess the pattern and severity of sleep disturbance and to gauge the role of sleep-related anxiety, dysfunctional beliefs about sleep, and presleep arousal in maintaining both primary and pain-related insomnia.

#### **Insomnia Severity Index (ISI<sup>31</sup>)**

The ISI is a 7-item scale validated with reference to the diagnostic criteria for primary insomnia in the DSM-IV.<sup>36</sup> Participants were instructed to respond to the questions based on their sleep in the last month. Each item is rated on a 5-point scale (0 = “Not at all,” 4 = “Extremely”) and summed to generate a total score that ranges from 0 to 28. A score of 15 or above identifies a diagnosis of clinical insomnia with excellent sensitivity (94%) and specificity (94%). The ISI has good levels of internal consistency (Cronbach  $\alpha = 0.76$  to 0.78; item-total  $r = 0.36$  to 0.67) and concurrent validity (correlation with sleep diary variables = 0.32 to 0.91; correlation with polysomnography variables = 0.07 to 0.45; correlation with clinician’s ratings = 0.50 to 0.71). In the present study, 6 additional questions were added to the end of the ISI asking the participants to report their (1) duration and (2) frequency of insomnia and their typical (3) sleep onset latency, (4) number and (5) duration of awakening after sleep onset and (6) total sleep time.

#### **Anxiety and Preoccupation About Sleep Questionnaire (APSQ<sup>37</sup>)**

The APSQ comprises 10 items measuring sleep-related anxiety, which were derived from insomnia patients’ statements concerning their insomnia (eg, “I worry about my loss of control over sleep”). Participants were asked to consider how they felt during the past month and rate their agreement to each item on a 10-point scale (1 to 10).

Responses are summed to provide a possible total score between 10 and 100, with a higher score indicating a higher level of sleep-related anxiety. The APSQ has demonstrated good internal consistency (Cronbach’s  $\alpha = 0.92$ ) and concurrent validity (correlation with the Pittsburgh Sleep Quality Index = 0.44 and with the Beck Anxiety Inventory = 0.37).

#### **Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS<sup>38,39</sup>)**

The DBAS is a measure of sleep-related beliefs and attitudes (eg, “I need 8 h of sleep to feel refreshed and function well during the day”). It contains 30 items that relate to expectations of required sleep, causal attributions and perceived consequences of insomnia, control and predictability of sleep, and beliefs about sleep-promoting practices. Participants were asked to rate their level of agreement with each statement between 0 “Strongly disagree” and 10 “Strongly agree.” A higher average score indicates a stronger presence of dysfunctional beliefs about sleep. The DBAS has demonstrated good internal consistency (Cronbach  $\alpha = 0.80$ ; average item-total  $r = 0.37$ ).

#### **Presleep Arousal Scale (PSAS<sup>40</sup>)**

The PSAS contains 16 items that measure the presence of cognitive (PSAS-C, eg, racing mind) and somatic (PSAS-S eg, muscle tension) arousal before falling asleep. Participants were asked to indicate on a 5-point scale (1 “Not at all” to 5 “Extremely”) how intensely they experienced each symptom. The item ratings are summed to provide a score for both cognitive and somatic arousal that ranges from 8 to 40; a higher score indicates a higher level of presleep arousal. The PSAS has demonstrated good internal consistency for both subscales (Cronbach  $\alpha$  for cognitive arousal = 0.67 to 0.88, and for somatic arousal = 0.79 to 0.84) and has shown good test-retest reliability over a 3-week period for both the cognitive ( $r = 0.72$ ) and somatic ( $r = 0.76$ ) subscales.<sup>40,41</sup>

### **Data Analysis**

#### **Between-group Comparisons**

We first examined if there were any significant differences in demographics between the *Pain-related Insomnia Group* and the *Primary Insomnia Group*. Given the unequal-n design, Mann-Whitney *U* tests were conducted for continuous variables [eg, age and body mass index (BMI)], whereas  $\chi^2$  analyses were conducted for categorical variables (eg, sex and ethnicity). Any demographic differences observed between the 2 groups were included as fixed factors (if the variable was categorical) or covariates (if the variable was continuous) in subsequent multivariate analyses of covariance (MANCOVA).

Three sets of MANCOVA were conducted to test whether the mean differences observed between the *Pain-related Insomnia Group* and the *Primary Insomnia Group* on a combination dependent variables occurred above chance. The first set of MANCOVA included the 7 sleep parameters measured as dependent variables (ie, insomnia duration and frequency, ISI, sleep onset latency, number and duration of wake after sleep onset, and total sleep time). The second set of MANCOVA included the 4 general psychological characteristics measured as dependent variables (ie, HADS-A, HADS-D, SHAI, and PSWQ). The third set of MANCOVA included the 4 cognitive-behavioral

processes measured as dependent variables (ie, APSQ, DBAS, PSAS-C, and PSAS-S).

### Regression Analysis

A hierarchical regression analysis was carried out to examine which of the variables of interest contributed to the prediction of insomnia severity, among the 133 participants in the Pain-related Insomnia Group. The predicted variable was insomnia severity (as measured with the ISI). Pain intensity (as measured with a 0 to 10 pain VAS) was forced in as the primary predictor of the model, followed by the general psychological variables (anxiety, depression, health anxiety, worry) as the second block of predictors and the insomnia-related cognitive-behavioral processes (sleep-related anxiety, sleep dysfunctional beliefs, and presleep cognitive, and somatic arousal) as the third block of predictors. To explore their relative roles in predicting insomnia severity, these predicting variables were selected into the model in a stepwise manner. Although this method has been criticized for being atheoretical, we consider this fitting for this exploratory analysis as a definitive model of pain-related insomnia is currently lacking.

## RESULTS

### Participant Characteristics

Characteristics of the participants are presented in Table 1. The 2 groups did not differ in terms of age, gender and ethnicity composition, educational level, and marital status. The mean age of the participants was 45 years. The majority of these individuals were female (76% to 81%), white (68% to 72%), and had received education below degree level (68% to 79%). Participants in the *PRIG* reported a mean pain severity of 6 out of a possible 10, which represents “worst possible pain.” The median pain duration reported by this group of patients was 96 months (ie, 8y).

Aside from the presence of chronic pain, significant between-group differences were observed for BMI ( $Z = -3.03$ ,  $P < 0.01$ ), employment ( $\chi^2 = 4.73$ ,  $P < 0.05$ ), and benefit ( $\chi^2 = 4.47$ ,  $P < 0.05$ ) status, such that the Pain-related Insomnia Group had a higher mean BMI and a higher percentage of participants who were out of employment and/or receiving benefits than the Primary Insomnia Group. Because of these observed differences, BMI was included in subsequent MANCOVA as a covariate, and employment and benefit status as additional independent variables.

### Pattern and Severity of Sleep Disturbance

Table 2 presents descriptive data on various sleep parameters differentiated by group, employment, and benefit status. Log10 transformation was used to correct excessive skewness and kurtosis in insomnia duration, sleep onset latency, and number and duration of awakenings after sleep onset. The first set of MANCOVA detected no significant main effect for group, BMI, employment, or benefit status. There was no significant interaction between and among all independent variables.

### General Psychological Variables

Table 3 presents descriptive data on the general psychological variables by group, employment or benefit status. The second set of MANCOVA demonstrated a

significant main effect for group (Wilks  $\lambda = 0.92$ ,  $F = 3.22$ ,  $df = 4, 155$ ,  $P < 0.05$ ,  $\eta^2 = 0.08$ ). Between-subjects tests indicated a significant main effect for group on the PSWQ [ $F(1, 158) = 8$ ,  $P < 0.01$ ], with the Primary Insomnia Group having a higher score than the Pain-related Insomnia Group. No significant main effect was detected for BMI, employment or benefit status. Whilst there was a significant interaction between group and benefit status (Wilks  $\lambda = 0.93$ ,  $F = 2.7$ ,  $df = 4, 155$ ,  $P < 0.05$ ,  $\eta^2 = 0.07$ ), between-subjects tests detected no specific effect for any of the psychological variables. No other significant interactions between and among the independent variables were detected.

### Insomnia-related Cognitive-behavioral Processes

Table 4 presents the descriptive data of various insomnia-related cognitive behavioral processes by group, employment, and benefit status. The third set of MANCOVA demonstrated a significant main effect for Group (Wilks  $\lambda = 0.91$ ,  $F = 3.65$ ,  $df = 4, 152$ ,  $P < 0.01$ ,  $\eta^2 = 0.09$ ). Between-subjects tests indicated that the effect of group was significant for the DBAS [ $F(1, 155) = 5.65$ ,  $P < 0.05$ ] and PSAS-C [ $F(1, 155) = 4.86$ ,  $P < 0.05$ ], with the Primary Insomnia Group scoring more highly on these measures than the Pain-related Insomnia Group. The main effect for Employment was also significant (Wilks  $\lambda = 0.94$ ,  $F = 2.64$ ,  $df = 4, 152$ ,  $P < 0.05$ ,  $\eta^2 = 0.07$ ). Between-subjects tests indicated specific effects for PSAS-S [ $F(1, 155) = 7.3$ ,  $P < 0.01$ ], with participants who were out of employment scoring more highly on the PSAS-S than those who were in employment. No significant main effect was detected for benefit status or BMI. There was no significant interaction between and among all independent variables.

### Regression Analysis

Table 5 summarizes the results of various hierarchical regression models built to predict the severity of pain-related insomnia. Pain intensity was entered into the equation as the primary predictor (see Model 1). Consistent with previous research findings,<sup>42</sup> pain intensity was found to be a significant predictor of insomnia severity, accounting for 11% of the variance. Next, depression was selected into the model (see Model 2). Independent of the contribution of pain intensity, depression explained an additional 12% of the total variance. Health anxiety was also selected into the model as a predictor of pain-related insomnia (see Model 3). Although the contribution was significant, the amount of variance explained by health anxiety was relatively small (3%) compared with those explained by pain intensity and depression. It became a nonsignificant predictor when presleep cognitive arousal was entered into the model to explain an additional 4% of the total variance (see Model 4). This suggests a possible overlap between the contributions of health anxiety and presleep cognitive arousal in predicting pain-related insomnia. A further hierarchical regression on insomnia severity was thus performed with pain intensity, depression and presleep cognitive arousal as the primary, secondary and tertiary predictors, respectively. The results of this model (Model 5) were similar to those of the previous model including health anxiety (Model 4), with the combination of all 3 predictors accounting for a total of 30% of the variance. On the basis of efficiency, Model 5 was considered the most fitting model.

**TABLE 1.** Participant Characteristics by Group

	Pain-related Insomnia (n = 137)	Primary Insomnia (n = 31)	Mann-Whitney U
Age (y)	46 (11.3)	44.7 (12.3)	Z = -0.52
Body mass index	27.8 (6.1)	24.1 (5)	Z = -3.03**
			$\chi^2$ (1, N = 168)
Sex (female %)	75.9	80.6	0.32
Ethnicity (white %)	72.3	67.7	0.25
Education (degree %)	21.2	32.3	1.74
Marital status (married %)	48.2	29	3.75
Employment status (unemployed or on sick leave %)	40.1	19.4	4.73*
Benefit status (receiving benefit %)	53.3	32.3	4.47*
Pain duration (mo)	125.3 (116.4)†	n/a	
Pain severity (0-10 VAS)	6.0 (2.4)	n/a	

Means and standard deviations in parentheses are presented, unless otherwise stated.

\*P < 0.05, \*\*P < 0.01. †Median = 96 months.

n/a indicates not applicable.

**DISCUSSION**

Direct comparisons of the characteristics of patients with primary and pain-related insomnia revealed no remarkable differences in their typical sleep patterns. Just like patients with primary insomnia, chronic pain patients with insomnia reported problems sleeping of equally long duration (> 90 mo), high frequency (5 to 6 nights/wk), and strong severity (mean ISI = 20, falling in the “severe clinical insomnia” range). As in primary insomnia, the profile of

pain-related insomnia was also characterized by extended sleep onset latency (> 1 h), long (> 1 h) and frequent (3 to 4 times) awakenings after sleep onset and short total sleep time (< 5 h). These findings complement previous polysomnography studies that recorded no significant difference in sleep architecture between the 2 types of insomnia.<sup>20,21</sup>

The 2 insomnia groups did not seem to differ from each other in terms of the psychological characteristics measured, with the exception that excessive worry seemed

**TABLE 2.** Means and Standard Deviations of Sleep Parameters by Group, Employment Status, and Benefit Status

	Pain-related Insomnia (n = 137)					Primary Insomnia (n = 31)				
	Group Total	Out of Employment		In Employment		Group Total	Out of Employment		In Employment	
		On Benefit (n = 48)	No Benefit (n = 7)	On Benefit (n = 25)	No Benefit (n = 57)		On Benefit (n = 6)	No Benefit (n = 0)	On Benefit (n = 4)	No Benefit (n = 21)
Insomnia duration (mo)*	92.6 (95.9)	82.2 (64.4)	161 (163.3)	125.4 (153.2)	80.1 (72.3)	98.5 (96.7)	70.8 (39.9)	n/a	96 (91.9)	105.6 (108.2)
Insomnia frequency (night/wk)	5.9 (1.5)	6.2 (1.4)	6 (1.7)	5.9 (1.5)	5.6 (1.6)	5.3 (1.8)	7 (0)	n/a	5.5 (1.9)	4.9 (1.8)
Insomnia severity (ISI)	20.5 (3.9)	21.8 (3.8)	20.2 (4)	20.8 (3.9)	19.2 (3.6)	19.5 (4.5)	23.4 (5.1)	n/a	22.8 (5.7)	18 (3.3)
Sleep onset latency (SOL; min)*	69.9 (61.6)	84.8 (72.7)	88.7 (72.3)	61.7 (51.5)	58.6 (51.8)	81.9 (74.2)	129 (101.5)	n/a	101.3 (67.5)	67 (66.1)
No. awakening (WASOn; time)*	3.5 (2.3)	3.6 (2.1)	3.4 (1.5)	3.2 (1.7)	3.5 (2.7)	2.8 (1.9)	2.6 (1.7)	n/a	2.5 (0.6)	2.9 (2.1)
Duration of awakening (WASOd; min)*	62.7 (51.8)	75.6 (60)	40.4 (43.5)	64.5 (55.5)	53.3 (40.9)	53 (50.7)	73 (96.2)	n/a	75 (57.4)	44.1 (33.2)
Total sleep time (TST; min)	287.5 (80.8)	254.4 (86.2)	277.5 (106.4)	292.6 (84.9)	314.7 (60.6)	294.5 (73.7)	252 (65.7)	n/a	300 (64.8)	303.6 (76.5)

Means are presented with standard deviations in parentheses.

\*Log10 transformed data were used for the actual analysis due to excessive skewness and kurtosis.

n/a indicates not applicable.

**TABLE 3.** Means and Standard Deviations of General Psychological Variables by Group, Employment Status, and Benefit Status

	Pain-related Insomnia (n = 137)					Primary Insomnia (n = 31)				
	Group Total	Unemployed		Employed		Group Total	Unemployed		Employed	
		On Benefit (n = 48)	No Benefit (n = 7)	On Benefit (n = 25)	No Benefit (n = 57)		On Benefit (n = 6)	No Benefit (n = 0)	On Benefit (n = 4)	No Benefit (n = 21)
Anxiety (HADS-A)	10.1 (3.8)	11.3 (3.7)	9.7 (2.8)	10.3 (3.7)	9.1 (3.9)	10.8 (4.3)	12.8 (3.6)	n/a	11 (5)	10.3 (4.3)
Depression (HADS-D)	8.8 (4.3)	10.1 (4)	11 (5.3)	9.1 (4.3)	7.2 (3.9)	7.4 (4.3)	12.2 (4.1)	n/a	10 (1.6)	5.8 (3.6)
Health Anxiety (SHAI)	15.4 (7.5)	17.9 (7.5)	17.7 (7.6)	15.4 (6.9)	13.1 (7.2)	13.4 (7.5)	16.6 (10.2)	n/a	11.5 (4.8)	13 (7.3)
Worry (PSWQ)	48.6 (14.6)	50.2 (14.3)	53.1 (17)	43.9 (13.3)	48.7 (15.1)	55.9 (14.6)	62.2 (7.9)	n/a	63.8 (13.3)	52.9 (15.4)

Means and standard deviations in parentheses are presented. HADS indicates Hospital Anxiety and Depression Scale; n/a, not applicable; PSWQ, Penn State Worry Questionnaire; SHAI, Short Health Anxiety Inventory.

to be a particular problem for patients with primary insomnia, rather than for those with pain-related insomnia. This is consistent with the cognitive conceptualizations of insomnia that emphasize the role of worry and negative thinking in manifesting problems sleeping.<sup>9-13,43</sup> Although excessive worry can be a by-product of sleeplessness, a number of experimental studies have found evidence indicating a causal role for worry—particularly during the presleep period—in delaying both subjective and objective sleep onset.<sup>44-48</sup> However, the difference observed between groups should not be interpreted as an absence of worry in pain-related insomnia, because the Pain-related Insomnia Group’s mean score on the PSWQ was above the clinical cutoff for generalized anxiety disorder (45).<sup>49</sup> As a group, patients with pain-related insomnia also had elevated scores above the suggested clinical thresholds on the HAD ( $\geq 8$ )<sup>50</sup> and SHAI ( $\geq 15$ ). These indicated concomitant mood and anxiety problems, which if not addressed properly may act as roadblocks to recovery. Indeed, evidence has emerged

from a recent large-scale longitudinal study that distress in the form of anxiety and depression significantly contributes to the prediction of insomnia maintenance at 1-year follow-up.<sup>51</sup>

The present study also examined the presence of sleep-related anxiety, dysfunctional beliefs about sleep, and presleep cognitive and somatic arousal in pain-related insomnia. These sleep-specific processes have been established in the primary insomnia literature as factors involved in the perpetuation of insomnia and thus form key treatment targets in CBT-I. In the present study, the Pain-related Insomnia Group seemed to match the Primary Insomnia Group in terms of their levels of sleep-related anxiety and somatic arousal during the presleep period. The Primary Insomnia Group, however, could be distinguished from the Pain-related Insomnia Group by their significantly stronger dysfunctional beliefs about sleep and higher levels of presleep cognitive arousal. The similarities between groups help explain the therapeutic benefits associated with the applica-

**TABLE 4.** Means and Standard Deviations of Insomnia-related Cognitive-behavioral Processes by Group, Employment Status, and Benefit Status

	Pain-related Insomnia (n = 137)					Primary Insomnia (n = 31)				
	Group Total	Out of Employment		In Employment		Group Total	Out of Employment		In Employment	
		On Benefit (n = 48)	No Benefit (n = 7)	On Benefit (n = 25)	No Benefit (n = 57)		On Benefit (n = 6)	No Benefit (n = 0)	On Benefit (n = 4)	No Benefit (n = 21)
Sleep-related anxiety (APSQ)	54.8 (23.1)	55.5 (23.3)	65.7 (17.4)	56.8 (22.8)	51.9 (23.7)	64.8 (16.9)	72 (22)	n/a	56.8 (9.1)	64.6 (16.8)
Dysfunctional beliefs about sleep (DBAS)	4 (1.5)	4.2 (1.6)	5 (1.6)	4.1 (1.7)	3.7 (1.3)	4.7 (1.1)	5.1 (1.2)	n/a	4.9 (0.7)	4.6 (1.1)
Presleep cognitive arousal (PSAS-C)	22.7 (7.4)	23.7 (7)	23.4 (5.3)	21.4 (7.8)	22.2 (7.7)	26.6 (6.8)	27.8 (10.3)	n/a	27.8 (6.5)	26.1 (6.2)
Presleep somatic arousal (PSAS-S)	17.4 (7)	20.6 (7.3)	20.7 (6.5)	16.8 (6.6)	14.7 (5.8)	16.3 (6.5)	20 (7.9)	n/a	14 (5.4)	15.9 (6.4)

Means are presented with standard deviations in parentheses. APSQ indicates Anxiety and Preoccupation about Sleep Questionnaire; DBAS, Dysfunctional Beliefs and Attitudes about Sleep Scale; n/a, not applicable; PSAS-C, Presleep Arousal Scale-cognitive; PSAS-S, Presleep Arousal Scale-somatic.

**TABLE 5.** Results of Hierarchical Regression Predicting Insomnia Severity Among Chronic Pain Patients With Sleep Problems

	Predicting Variable	R <sup>2</sup>	Adjusted R <sup>2</sup>	R <sup>2</sup> Change	F	P	B	SE	β	t	P
Model 1	Pain intensity	0.11	0.1	0.11	15.84	< 0.001	0.54	0.14	0.33	3.98	< 0.001
Model 2	Pain intensity	0.11	0.1				0.41	0.13	0.25	3.21	< 0.01
	Depression	0.23	0.22	0.12	19.5	< 0.001	0.33	0.07	0.36	4.57	< 0.001
Model 3	Pain intensity	0.11	0.1				0.37	0.13	0.23	2.89	< 0.01
	Depression	0.23	0.22				0.26	0.08	0.29	3.42	< 0.01
	Health anxiety	0.26	0.24	0.03	14.94	< 0.001	0.09	0.04	0.18	2.16	< 0.05
Model 4	Pain intensity	0.11	0.1				0.37	0.13	0.23	2.98	< 0.01
	Depression	0.23	0.22				0.25	0.08	0.27	3.29	< 0.01
	Health anxiety	0.26	0.24				0.04	0.05	0.08	0.89	0.374
	Presleep cognitive arousal	0.30	0.28	0.04	13.71	< 0.001	0.12	0.04	0.23	2.78	< 0.01
Model 5	Pain intensity	0.11	0.1				0.38	0.12	0.24	3.16	< 0.01
	Depression	0.23	0.22				0.26	0.07	0.29	3.83	< 0.001
	Presleep cognitive arousal	0.30	0.28	0.07	18.67	< 0.001	0.14	0.04	0.27	3.59	< 0.001

tion of CBT-I to pain-related insomnia.<sup>52–55</sup> Although the differences between groups do not diminish the importance of dysfunctional sleep beliefs and presleep cognitive arousal in pain-related insomnia, they point to areas where our understanding of pain-related insomnia needs to be refined.

An inspection of the individual items of the DBAS suggests that the relatively lower scores among chronic pain patients with insomnia may be attributable to the context. For example, item 14 of the DBAS is a statement concerning the assumed cause of insomnia: “I feel that insomnia is basically the result of aging and there isn’t much that can be done about this problem.” This item is likely to be less relevant to chronic pain patients because many of them hold the beliefs that their insomnia is a result of their pain and that it will be relieved when the pain is gone.<sup>24,56</sup> Clinical experience also suggests that prominent sleep beliefs among chronic pain patients tend to focus on the interaction between sleep and pain. For instance, many patients firmly believe that when they are in pain, it is simply impossible for them to get comfortable and go to sleep. Potentially, it is of research and clinical interest to develop a pain-specific DBAS to capture beliefs pain patients may have developed about the pain-sleep interaction. Similarly, the relatively lower scores on the PSAS-C may be attributable to the fact that thoughts preoccupying the minds of chronic pain patients are mostly concerned with pain,<sup>57</sup> which is not a subject featured in any of the PSAS-C items. Consistent with this idea, a previous study has shown that pain-related thoughts are more frequently reported by chronic pain patients during the presleep period than other thoughts such as those about sleep, about what happened during the day, and about the immediate environment.<sup>29</sup>

Participants in this study were recruited from clinics and general practices located within the same catchment area. We did not anticipate the 2 groups of patients to be different in demographics. Interestingly though, we found that significantly more patients in the Pain-Related Insomnia Group were out of employment (40.1%) or on benefits (53.3%) relative to the Primary Insomnia Group (19.4%; 32.3%). Patients with pain-related insomnia also had a significantly higher mean BMI (27.8) compared with those with primary insomnia (24.1). Results of the MANCOVA indicated an effect of people’s employment status on their reported levels of presleep somatic arousal, such that those individuals who were out of employment were more likely to report higher levels of presleep somatic tension compared with those who were in employment. This

finding reflects the potential impact of our socioeconomic environment on our well-being and it seems sensible for clinicians to bear these demographic differences in mind when devising treatments for patients suffering from both problems.

Regression analyses were conducted to identify potential predictors of pain-related insomnia. The results indicated that, while pain intensity did account for a certain amount of variance, depression and presleep cognitive arousal also significantly contributed to the manifestation of insomnia in the current sample. These findings corroborate earlier reports by our group and other investigators noting the importance of mood,<sup>58</sup> presleep rumination,<sup>29</sup> and affective responses to pain<sup>22</sup> in pain-related insomnia. Although the direct application of CBT-I has shown efficacy in reducing insomnia symptoms in the context of chronic pain, optimal treatment for pain-related insomnia may require simultaneous effort to address all factors involved. Our group is currently evaluating the utility of a hybrid treatment for pain-related insomnia that includes components to target factors underpinning sleeplessness, low mood, and pain.

These regression results also highlight the psychological avenues through which pain and sleep may reinforce each other. The reciprocal links between pain and depression<sup>59</sup> and between sleep and depression<sup>60</sup> have separately attracted a reasonable amount of research. However, clinical and epidemiological observations suggest that these entities rarely operate in isolation.<sup>61,62</sup> Bolstered by recent findings from fMRI and experimental studies that the same neuroendocrine circuitry is involved in the experience of pain and depressed mood, as well as the regulation of sleep,<sup>63–65</sup> future investigations—particularly those that use a longitudinal design—should consider examining the tripartite interaction.

Methodologically, we recruited more chronic pain patients than primary insomnia patients in this study, as we were interested in conducting an adequately powered regression analysis to identify psychological predictors of the pain-related insomnia. One potential issue with the unequal-n design is the possibility of unequal (co)variance across groups, but nonparametric tests were used to detect between-groups differences and in each set of MANCOVA, the Box’s test of equality of covariance matrices of the dependent variables was non-significant, indicating that the assumption of homoscedasticity was not violated. The data collected in this study relied on the participants’ self-report and were therefore subject to possible reporting or recall

bias. Although the insomnia literature has suggested that subjective sleep reports may be systematically different from objective sleep records provided by sleep-estimating technologies (eg, actigraphy or polysomnography),<sup>66</sup> findings of the current study corroborated those of previous studies using polysomnography in identifying a comparable sleep disturbance profile between patients with primary and pain-related insomnia. It is thus concluded with some confidence that, at the macro-level, the 2 types of insomnia are broadly similar in presentation.

This said, future studies should pay attention to the effect of pain on sleep at the micro-level.

Using a more sensitive measure of sleep disruption, some researchers have identified an increased rate of cyclical alternating pattern (CAP) of EEG activity to be indicative of a worse quality of sleep in patients with fibromyalgia.<sup>67</sup> CAP corresponds to a prolonged oscillation of the arousal level between 2 reciprocal functional states termed phase A (greater arousal) and phase B (lesser arousal). It is thought to represent a condition of instability that manifests the brain's fatigue in preserving and regulating the macro-structure of sleep. CAP has been proposed to be a unique marker of sleep disturbance in fibromyalgia. However, the specificity of CAP to chronic pain conditions, including fibromyalgia, awaits empirical examination.

In summary, the present study found no evidence to suggest that patients suffering from pain-related insomnia differ from patients with primary insomnia in their typical sleep patterns. The 2 groups were largely similar in terms of their psychological characteristics, except that patients with primary insomnia were distinguishable from those with pain-related insomnia by their greater tendency to worry. Cognitive-behavioral processes known to be involved in the maintenance of primary insomnia seem to also operate in insomnia linked to chronic pain. In particular, patients with pain-related insomnia reported levels of sleep-related anxiety and presleep somatic arousal that were comparable to those reported by patients with primary insomnia. These overlaps help explain the therapeutic benefits associated with the direct application of CBT-I for the treatment of pain-related insomnia. There were, however, differences between the 2 insomnia groups in terms of their subscription to dysfunctional sleep beliefs and the presence of presleep cognitive arousal. Finally, findings of the present study also suggest that, in addition to pain, negative mood and a racing mind during the presleep period are also significant predictors of insomnia severity among chronic pain patients. Given the intricate links between the pain, mood and sleep, it is suggested that hybrid treatments that seek to simultaneously address factors across all 3 domains may represent more effective treatments than 1-dimensional interventions.

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