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**Insomnia In Chronic Pain Patients With and Without
Major Depressive Disorder**

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Abstract

Insomnia and major depression are both common problems among people with chronic pain. Although each has been studied quite extensively in this population, they have seldom been considered together. Thus, the goal of the present research was to compare patients with chronic pain who either did or did not meet criteria for major depressive disorder, with a particular emphasis on measures of sleep and sleep disturbance. Thirty-three patients with chronic pain and comorbid major depression and 27 chronic pain patients without comorbid major depression completed structured diagnostic interviews on sleep and mood, and completed retrospective and 4-day diary measures of sleep, sleep-related behaviours, sleep-related cognitions, pain, and mood. The measures of sleep-related variables were selected because of their relevance to a cognitive-behavioural model of insomnia.

A diagnosis of insomnia was highly prevalent in both groups, with fully 53 of 60 (88.3%) participants meeting DSM-IV criteria. Participants with major depression reported significantly higher levels of pain and more dysfunctional attitudes and behaviours related to sleep. They were also more likely to have sleep disorders other than insomnia. However, specific sleep parameters such as sleep onset latency, time awake after sleep onset, sleep efficiency, and total sleep time did not differ between the groups, whether they were assessed retrospectively or with daily diaries. Insomnia symptoms were highly associated with pain severity among participants with major depression; however, the majority of pain, sleep, and mood-related correlates of insomnia severity did not differ significantly between the groups. Partial correlation coefficients, controlling for pain severity, substantially decreased the associations between depression

and cognitive-behavioural variables related to sleep, thus suggesting that pain may be more important than depression in the etiology of insomnia. That is, chronic pain itself may disturb sleep so extensively that comorbid depression has little incremental effect. The results are discussed in terms of the potential applicability of cognitive-behavioural interventions for insomnia in chronic pain patients with comorbid major depressive disorder.

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Study Overview

Chronic pain, depression, and insomnia are all important public health problems that affect sizeable portions of the population. In addition, these problems frequently occur together. Psychologists have been active contributors to the conceptualization and treatment of each of these disorders, and each is amenable to psychological intervention using methods that have been developed within a cognitive-behavioural framework. However, there is a paucity of research that has considered the conjoint influence of these problems, by investigating the cognitive and behavioural dimensions of insomnia among chronic pain patients with comorbid major depression. This is an important clinical consideration, because cognitive-behavioural treatments for insomnia appear to be effective in helping to alleviate the sleep disturbance in people who have chronic pain. As with other applications of these nonpharmacological interventions for sleep, however, patients who are concurrently depressed have generally been excluded from research (Currie, Wilson, Pontefract, & deLaplante, 2000; Smith & Perlis, 2006; Stepanski & Rybarczyk, 2006). Nevertheless, there is a small body of evidence to suggest that cognitive-behavioural therapy might indeed be an appropriate treatment for persistent insomnia, even when it is complicated with comorbid depression. However, additional research is necessary before such treatment recommendations can be assessed in clinical trials and implemented responsibly -- research that provides a solid empirical basis for the appropriateness of the various cognitive and behavioural characteristics that are the targets of intervention in this type of therapy (Smith, Huang, & Manber, 2005; Smith & Perlis, 2006; Stepanski & Rybarczyk, 2006).

The current study involved a detailed examination of retrospective and daily diary indices of the self-reported sleep characteristics of chronic pain patients in conjunction with structured diagnostic interviews to compare participants with and without comorbid major depression. Although these characteristics have been examined before in the chronic pain literature, the available research has largely been restricted to examining retrospective reports of 'typical' sleep, rather than with daily monitoring. Moreover, there has been little attempt to consider the constructs that are usually the targets of treatment in cognitive-behavioural therapy for insomnia. Therefore, in the present study, cross-sectional analyses were conducted on participants' sleep parameters, sleep-related behaviours, and sleep-related thoughts, attitudes, and beliefs, using data derived from both retrospective and daily diary measures.

Three primary hypotheses were evaluated: (1) In keeping with previous research (Benjamin, Barnes, Berger, Clarke, & Jeacock, 1988; Currie & Wang, 2004; Elliott, Renier, & Palcher, 2003; Haley, Turner, & Romano, 1985; Haythornthwaite, Sieber, & Kerns, 1991), it was hypothesized that chronic pain patients with comorbid major depression would report significantly higher levels of pain and disability compared to patients without major depression. (2) It was hypothesized that pain patients with major depression would report more problematic sleep parameters and sleep-related behaviours than pain patients without comorbid major depression (Lacks & Rotert, 1986; Woodley & Smith, 2006). (3) Consistent with the previous hypothesis and the cognitive-behavioural conceptualization of insomnia, it was expected that pain patients with major depression would endorse significantly more maladaptive sleep-related cognitions than

their nondepressed counterparts (e.g., Smith et al., 2005; Smith & Perlis, 2006; Woodley & Smith, 2006).

Finally, a series of exploratory analyses were conducted on mood-related variables. This was performed in order to verify the validity of the mood-related diagnoses. Next, the correlates of global insomnia severity were examined within and between the groups. Finally, the association between mood-related variables and the targets of cognitive-behavioural therapy for insomnia were explored. These were undertaken due to the paucity of data regarding the insomnia experienced by individuals with chronic pain and comorbid major depression, and the tendency for these patients to be excluded from research (e.g., Currie et al., 2000; see Smith & Perlis, 2006, and Setpanski & Rybarczyk, 2006, for recent reviews).

Chronic Pain

Chronic pain is defined by the International Association for the Study of Pain (IASP) as pain that persists for more than six months, irrespective of medical treatment (Merskey & Bogduk, 1994). This definition is commonly used because a six-month period is thought to allow adequate time for acute tissue damage to heal after an injury. As opposed to acute pain, which serves an adaptive function in attracting attention to injury, chronic pain is a complex syndrome composed of a variable constellation of psychological and behavioural symptoms, in addition to overt physical pathology, if any (Verhaak, Kerssens, Dekker, Sorbi, & Bensing, 1998). Chronic pain is associated with many diverse diagnoses and conditions, such as physical trauma, back, neck and musculoskeletal problems, arthritis/rheumatic diseases, migraine headaches, and temporomandibular joint dysfunction. For some chronic pain conditions, such as

fibromyalgia (a condition characterised by widespread muscular tender points and fatigue; Wolfe et al., 1990), the underlying medical etiology is unknown or unclear (Kerns, 1996). These facts notwithstanding, some researchers have argued that the behavioural dimension of diverse pain conditions can be studied together because they share common psychological features (Fields, 1987; Madland, Feinmann, & Newman, 2000; Townsend, Sletten, Bruce, Rome, Luedtke, & Hodgson, 2005; Turk & Rudy, 1990).

Although epidemiological studies have yielded disparate results, chronic pain clearly affects a significant minority of the general population. A survey of 2,184 Saskatchewan adults revealed that 28.4% experienced back pain at the time of the survey (Cassidy, Carroll, & Cote, 1998), while 22.2% experienced neck pain (Cote, Cassidy, & Carroll, 1998). Millar (1996) reported that 3.9 million, or 17% of Canadian adults, experience chronic pain; of these, 35% report pain that is significant enough to cause limitations in daily activities. Moulin, Clark, Speechley, and Morley-Forster (2002) conducted a stratified random telephone survey of 2,012 Canadian adults, and found that 29% suffered from chronic pain of six or more months in duration. Using data from the Canadian Community Health Survey ($n = 115,071$), Patten et al. (2005) reported prevalence estimates of 18.8% for chronic back problems, and 16.7% for arthritis or rheumatism.

Studies outside of Canada have reported similar findings. Bonica (1990) estimated that up to 30% of adults in the United States suffer from some form of chronic pain; similarly, Latham and Davis (1994) estimated that 40 to 70 million Americans experience chronic pain. Von Korff et al. (2005) examined a probability sample of 5,692 American

adults and found that 19% reported chronic spinal pain within the past year. Data from European countries are comparable or higher; in these cases, several studies have defined chronic pain as pain persisting for three or more months. For instance, Picavet and Schouten (2003) conducted a postal survey of 3,664 members of the Dutch population and estimated that 44.4% reported some form of musculoskeletal pain of three or more months' duration within the past year. In a survey of 11,797 female primary care patients in the UK, Smith, Elliott, & Hannaford (2004) reported that 38% of respondents experienced some form of chronic pain of that duration at the time of the study. A randomized postal survey of 4,611 individuals conducted by Smith, Elliott, et al. (2001) revealed that 20.4% of UK respondents had either 'significant' or 'severe' pain of three or more months' duration. In a Norwegian population-based survey, 24.4% of respondents had experienced chronic pain for three or more months (Rustoen et al., 2004). A slightly smaller percentage (14.3%) of 4,542 Finnish survey respondents reported similarly (Mantyselka, Turunen, Ahonen, & Kumpusalo, 2003). When chronic pain is defined as pain persisting for six months or more, prevalence estimates are comparable. Gureje, Simon, and Von Korff (2001) analyzed interview data from 3,197 primary care patients from 15 health centres in 14 European countries at baseline and 12 months later. They estimated that 22.7% of respondents experienced chronic pain at baseline; of these, 49.1% continued to experience pain at follow-up. Ohayon & Schatzberg (2003) conducted a telephone survey of 18,980 individuals from five European countries and found that 17% reported one or more chronic pain conditions lasting six months or more. Nineteen percent of 10,066 Danish respondents also reported pain of more than six months' duration (Eriksen, Jensen, Sjogren, Ekholm, & Rasmussen,

2003). More recently, Breivik, Collett, Ventafridda, Cohen, and Gallacher (2006) conducted a large-scale telephone survey of 46,394 individuals from 15 European countries and Israel and found that 19% reported suffering from a pain condition of six or more months' duration.

Taken together, these estimates are generally consistent with earlier research (Seers, 1992), where it was estimated that 25% to 30% of the general populations of industrialized nations are affected by chronic pain. For instance, Verhaak et al. (1998) reviewed 15 epidemiological studies of chronic pain and obtained prevalence estimates ranging from 2% to 40%, with a median point prevalence of 15%.

In addition to its physical and emotional costs, chronic pain poses a substantial economic burden as well. In fact, it has been reported that chronic pain costs \$40 billion (Aronoff, Evans, & Enders, 1983) to \$215 billion dollars (US) per year (American Academy of Orthopaedic Surgeons, 1999; US Census Bureau, 1996; US National Research Council, 2001) in terms of health care and related costs, resulting in over 400 million lost workdays (Bonica, 1990). Recent population-based research from Australia suggests that chronic pain contributes to 9.9 million workdays lost per year due to absenteeism, and 36.5 million workdays lost due to reduced effectiveness associated with chronic pain. The authors estimated that this results in an annual cost of \$5.1 billion AUD annually (van Leeuwen, Blyth, March, Nicholas, & Cousins, 2006). Chronic pain patients also use health care services two to three times as frequently as individuals without chronic pain (Blyth, March, Brnabic, & Cousins, 2004; Eriksen, Sjogren, Ekholm, & Rasmussen, 2004; Millar, 1996; Von Korff, Dworkin, & Le Reshe, 1990). Research has also indicated that chronic pain sufferers are more likely to be female, older, and to have

lower incomes (Eriksen, Ekholm, Sjogren, & Rasmussen, 2004; Gatchel, Polatin, & Mayer, 1995; Magni, Caldieron, Rigatti-Luchini, & Merskey, 1990; Millar, 1996; Moulin et al., 2002; Saastamoinen, Leino-Arjas, Laaksonen, & Lahelma, 2005).

Treatment of chronic pain. A variety of methods have been used to treat chronic pain. Although surgery is sometimes performed when a clearly correctable medical problem is identified, many individuals have to cope with their pain on a long-term basis. In general, interventions that are intended to help in this context can be characterized as either pharmacological or nonpharmacological in nature. Pharmacological interventions, such as analgesics, anti-inflammatory preparations, and antidepressant drugs are used to treat pain, inflammation and the emotional distress that frequently accompanies chronic pain. For many individuals, however, pharmacological treatments provide only a partial solution to their problem. In addition, some types of medications carry health risks associated with the development of tolerance, dependence, and cumulative side effects (Aronoff & Evans, 1992).

Nonpharmacological therapies for chronic pain have focused on physical or behavioural (e.g., relaxation training, biofeedback, physiotherapy) as well as psychological (e.g., cognitive behavioural therapy) factors, with mixed empirical support as to their efficacy as stand-alone treatments (e.g., Schoicket, Bertelson, & Lacks, 1988; see Cohen & Campbell, 1996; Scheer, Watanabe, & Radack, 1997, reviews). In light of these findings, and the fact that pain appears to be a multidimensional construct with a variety of physical, psychological and social sequelae (Turk & Flor, 1999), multidisciplinary pain management clinics are currently the dominant approach to chronic pain treatment (Flor, Fydrich, & Turk, 1992; Guzman et al., 2001; Keefe,

Rumble, Scipio, Giordano, & Perri, 2004; McCracken & Gross, 1998; Scheer et al., 1997; Spinhoven et al., 2004; Sullivan, Reesor, Mikail, & Fisher, 1992; Turk, 1996b). In such clinics, patients are evaluated and treated by professionals from a variety of specialties (e.g., physiotherapy, psychology, physiatry). Implicit in treatment of this kind is the notion that psychosocial variables are critical in a patient's rehabilitation and resumption of regular activities (Keefe et al., 2004; Sullivan et al., 1992; Spinhoven et al., 2004). Multidimensional pain management clinics have been shown to be effective in the treatment of chronic pain conditions, particularly in terms of decreased pain, improved mood, resumption of activities, and decreased use of medication and health care services (Burns, Kubilus, Bruehl, Harden, & Lofland, 2003; Douglas, Graham, Anderson, & Rogerson, 2004; Flor et al., 1992; Guzman et al., 2001; Jensen, Turner, & Romano, 2001; McCracken & Gross, 1998; Scheer et al., 1997; Spinhoven et al., 2004; Turk, 1996b; Turk & Okifuji, 2002; Vowles, Gross, & Sorrell, 2004).

Gate Control Theory of Pain

The importance of psychological contributions to pain management has been emphasized in the influential 'gate control' theory of pain. Originally proposed by Melzack and Wall (1965), the gate control theory was developed to account for the diverse range of pain conditions that is evident clinically, such as the occurrence of pain without injury, or injury without pain (Gatchel, 1999; Horn & Munafo, 1997). The gate control theory revolutionized the field of pain research and has become the most important psychophysiological model within the field (Melzack & Katz, 2006; Robinson & Riley, 1999a; Turk & Flor, 1999).

The gate control theory postulates a relationship between physiological and psychological factors in the experience of pain. According to this theory, there is presumed to be a gating mechanism in the dorsal horn of the substantia gelatinosa of the spinal cord that modulates (inhibits or facilitates) nerve impulses from the body to the central nervous system (CNS) to determine the nature of the pain experience. This modulation is influenced by both ascending physiological activity from the peripheral nerves (e.g., tissue damage at the injury site) and by descending psychological factors (e.g., stress) reflected in the CNS. Due to this modulation at the site of the gating mechanism, there is not necessarily a direct relationship between peripheral tissue damage and the experience of pain (Horn & Munafo, 1997; Melzack & Casey, 1968; Melzack & Katz, 2006; Melzack & Wall, 1965; Turk & Flor, 1999). The gate control theory was revolutionary in that it went counter to the previously dominant belief that pain was either somatic or psychogenic in nature, rather than the product of interacting central and peripheral nervous system activities (Melzack & Katz, 2006; Turk & Flor, 1999).

In peripheral terms, the balance of activity of excitatory (large-diameter, A-beta) and inhibitory (small-diameter, A-delta and C) nerve fibres in the dorsal horn determines what peripheral information arrives at the gating mechanism. Pain modulation is also affected by psychological factors such as affect, learning, cognition, and behaviour, which are represented by neural impulses descending from the brain to the gating mechanism. These factors are thought to be subsumed under the body-self neuromatrix, a widely-distributed neural network that integrates somatosensory, limbic, and thalamocortical contributions to the pain experience (Melzack, 1999, 2001; Melzack &

Wall, 1965). These biological systems are viewed as subservient to the sensory-discriminative, affective-motivational, and evaluative-cognitive dimensions of the pain experience (Melzack, 1999, 2001; Melzack & Katz, 2006).

Thus, according to gate control theory, pain, particularly in its chronic form, is a multi-dimensional psychological experience. It is the result of the integration of ongoing perceptual processes, and is affected by psychological factors such as learning and memory in addition to peripheral tissue injury (Horn & Munafo, 1997; Keefe, Holzberg, & Beaupre, 1996; Melzack, 1999, 2001; Melzack & Katz, 2006; Melzack & Wall, 1965; Robinson & Riley, 1999a).

Psychological Conceptualizations of Chronic Pain

In acknowledging the role of psychological factors in the modulation of the pain experience, the gate control theory contributed significantly to psychology's involvement in the field of pain research and treatment. In fact, behavioural and cognitive psychotherapies have emerged as important approaches to the conceptualization and treatment of chronic pain.

Behavioural perspective. Described principally by Fordyce (1976, 1996), the behavioural or operant perspective emphasizes that the phenomenon of pain consists not only of the patient's subjective experience, but also of the patient's wide array of pain-related behaviours. These pain-related behaviours range from the general (e.g., level of functioning) to the specific (e.g., overt expressions of pain, medication usage).

Although operant factors are not implicated in the initial development of pain, the behavioural perspective contends that chronic pain problems develop when behaviours associated with acute pain (e.g., limping, complaining, inactivity, medication-seeking)

eventually come under the control of external contingencies or reinforcements from others (e.g., spousal or caregiver attention) or from the environment (e.g., time off from work). Accordingly, the influence of biomedical factors is thought to dissipate over time, to the extent that chronic pain conditions may persist well after the initial injury has healed, and for reasons other than the initial etiology (Fordyce, Shelton, & Dundore, 1982). Furthermore, in addition to receiving positive reinforcement for pain-related behaviours, patients may fail to receive reinforcement for 'well' or functional behaviours, such as physical activity, which can lead to muscular deconditioning that actually exacerbates the pain condition.

Treatments based on this model focus on pain-related behaviours, as opposed to the alteration of nociception *per se*, although the latter may occur nevertheless (Turk & Flor, 1999). Increased independence, enhanced functioning and rehabilitation are the ultimate goals of behavioural treatment, and these are achieved through the extinction of pain-related behaviours and an increase in adaptive or functional responses (Fordyce, 1976; Fordyce et al., 1982; Horn & Munafo, 1997; Turk & Flor, 1999). In general, empirical investigations have demonstrated the effectiveness of behavioural interventions within the milieu of multidisciplinary pain clinics (e.g., Block, 1982; Flor et al., 1992).

Cognitive perspective. According to the cognitive perspective, maladaptive cognitions influence the nature of the pain experience and serve to exacerbate and maintain pain, pain-related behaviours, and accompanying affective distress (e.g., Turk, 1986; 1996a). Consistent with the behavioural perspective, cognitive theories of pain assume that learning contributes significantly to the development of chronic pain

conditions (Horn & Munafo, 1997); similarly, biomedical factors are viewed as exerting a diminishing influence over time (Turk, 1996a; Turk & Flor, 1999; Turk & Rudy, 1986).

A variety of cognitive factors are hypothesized to moderate the relationship between nociception and the subjective pain experience. Negative cognitive schemata, decreased perceptions of self-efficacy, a perception of the self as disabled, and idiosyncratic beliefs about pain, one's condition, and one's ability to function in the face of pain, have been implicated as moderators of pain (see Turk, 1996a; Turk & Flor, 1999; Turk & Rudy, 1986; Turner, Ersek, & Kemp, 2005). Other relevant factors include erroneous beliefs about the controllability of pain, catastrophizing, fear of pain, locus of control, coping style, and attentional hypervigilance to physical symptoms (e.g., Arnstein, Caudill, Mandle, Norris, & Beasley, 1999; Burns et al., 2003; Douglas et al., 2004; Geisser, Robinson, Keefe, & Weiner, 1994; Jensen, Turner, & Romano, 1991, 1992; Lame, Peters, Vlaeyen, Kleef, & Patijn, 2005; Peters, Vlaeyen, & Weber, 2005; Turk & Fernandez, 1991; Turner et al., 2005; Turner, Jensen, & Romano, 2000; see Asmundson, Norton, & Norton, 1999; Boothby, Thorn, Stroud, & Jensen, 1999; Sullivan et al., 2001; Turk, 1996a, Turk & Rudy, 1992, for reviews). Indeed, some of the recent directions in treatment research include exposure therapies for feared activities (Vlaeyen, de Jong, Geilen, Heuts, & van Breukelen, 2001), attention management strategies (Morley, Shapiro, & Biggs, 2004), and acceptance-based treatment (McCracken & Eccleston, 2006; McCracken, Vowles, & Eccleston, 2005).

Cognitive-behavioural perspective. Although behavioural and cognitive perspectives on chronic pain are conceptually distinct, in practice they tend to be integrated with one another. The integration of cognitive and behavioural approaches has

become extremely influential in the field of chronic pain research and treatment (e.g., Flor et al., 1992; Spinhoven et al., 2004; Turk & Burwinkle, 2005; Turk & Okifuji, 2002; Wells-Federman, Arnstein, & Caudill-Slosberg, 2003). In this model, cognitive factors are acknowledged as contributors to pain modulation and affective distress; however, the contribution of behavioural factors in the development and maintenance of chronic pain and pain-related behaviours are also a focus of attention (e.g., Spinhoven et al., 2004; Turk & Burwinkle, 2005; Turk & Okifuji, 2002; Turk & Rudy, 1986).

Cognitive-behavioural therapy (CBT) is a central component of multidisciplinary pain clinics (e.g., Burns et al., 2003; Douglas et al., 2004; Evers, Kraaimaat, van Riel, & de Jong, 2002; Flor et al., 1992; Jensen et al., 2001; Lame et al., 2005; McCracken et al., 2005). CBT incorporates physical interventions (e.g., relaxation training or biofeedback to decrease muscle tension or anxiety; Smith & Perlis, 2006) with psychological ones (e.g., cognitive restructuring). The ultimate goals of CBT for chronic pain are to foster functional cognitions (e.g., beliefs, assumptions) and behaviours by addressing issues such as self-efficacy, coping strategies, and by altering beliefs so that patients come to view their situation as manageable. Patients are taught to monitor their thoughts, emotions, and behaviours and to note how these relate to pain, emotional distress, and psychosocial difficulties. In addition, CBT aids patients in the development of adaptive ways of thinking, feeling, and behaving after treatment has been completed (e.g., Bradley, 1996; Burns et al., 2003; Douglas et al., 2004; Keefe et al., 2004; McCracken et al., 2005; Turk & Fernandez, 1991; Turk & Okifuji, 2002).

Chronic Pain and Major Depression

Prevalence and correlates of major depression in chronic pain. Another important psychological dimension of the chronic pain experience is evident in the fact that many individuals with chronic pain develop clinically significant problems with depression. Several recent epidemiological studies have highlighted the frequent co-occurrence of chronic pain and major depression. Arnow et al. (2006) surveyed 5,808 primary care patients and found that significantly more patients with major depression reported suffering from chronic pain (66%) compared to individuals without major depression (43%). However, the work of Arnow et al. (2006) relied upon self-report data (collected via postal survey) in the diagnosis of major depression and chronic pain. Currie and Wang (2004) analyzed telephone interviews from 118,533 Canadian households as part of Statistics Canada's National Population Health Survey. Currie and Wang (2004) used the Short Form of the World Health Organization's Composite International Diagnostic Interview (CIDI), and reported that the overall prevalence of chronic back pain was 9%. However, the prevalence of major depression among individuals without chronic back pain was estimated to be 5.9%, whereas a significantly higher percentage (19.8%) of participants with chronic back pain were estimated to suffer from comorbid major depression. Currie and Wang (2004) observed a positive linear relationship between estimates of major depression and pain severity. In regression analyses, chronic back pain was the strongest predictor of major depression.

McWilliams, Cox, & Enns (2003) analyzed data from 5,877 respondents from the United States' National Comorbidity Survey, also using the CIDI. They found that 6.49% of respondents reported some severe form of arthritis, rheumatism, or other bone or joint

disease. Of these, 20.2% received a diagnosis of major depression, while 9.3% of participants without these forms of pain received a diagnosis of major depression. Ohayon (2004) and Ohayon and Schatzberg (2003) evaluated telephone interview data from randomized samples drawn from 5 European countries and found that, of the 4% reporting symptoms consistent with a diagnosis of major depression, 43.4% also had a concurrent chronic painful physical condition. Von Korff et al. (2005) also examined data from the National Comorbidity Survey and reported that 19% of participants reported some form of chronic spinal pain within the previous 12 months, and 29.3% reported a lifetime prevalence. However, in this case, chronic spinal pain was loosely self-defined by the respondent. Of those reporting chronic spinal pain within the previous year, 12.6% also had concurrent major depression. In a large-scale telephone survey of 46,394 individuals in 15 European countries and Israel, Breivik et al. (2006) reported that 19% of their sample suffered from chronic pain for six months or more; of these, 21% reported having been diagnosed with major depression after the development of chronic pain.

In addition to comorbidity studies within the general population, the prevalence of depressive disorders among pain clinic patients has also been the subject of a substantial body of research. Table 1 summarizes the results of 39 studies published within the past two and a half decades that used face-to-face structured diagnostic interviews in conjunction with criterion-based diagnostic systems (i.e., Diagnostic and statistical manual of mental disorders [DSM] or Research Diagnostic Criteria [RDC]) to assess major depression in chronic pain clinic patients. As is evident in this table, there are two main conclusions that can be drawn. First, the overall prevalence of major depression is high, as reflected in the fact that, across all studies, the average prevalence was 33.6 %.

Table 1. Major Depressive Disorder (MDD) Among Chronic Pain Clinic Patients

Study	n	Type of Pain	% Current MDD
Atkinson et al. (1988)	32	Low back	15.6
Atkinson et al. (1991)	97	Low back	21.6
Atkinson et al. (1996)	52	Mixed sites	44.2
Benjamin et al (1988)	106	Mixed sites	33.0
Bouckoms et al. (1988)	62	Mixed sites	24.2
Davidson et al. (1985)	57	Mixed sites	42.1
Elliott et al. (2003)	242	Mixed sites	52.0
Fishbain et al. (1986)	283	Low back	4.6
France et al. (1984)	42	Low back	52.4
France, Houpt, et al. (1986)	80	Low back	21.3
Frank et al. (1988)	137	Rheumatoid Arthritis	17.0
Geisser et al. (1997)*	132	Mixed sites	33.3
Geisser et al. (2000)*	211	Mixed sites	29.9
Goldenberg (1986)	82	Fibromyalgia	13.4
Haley et al. (1985)	63	Mixed sites	49.2
Haythornthwaite, Sieber, & Kerns (1991)	91	Mixed sites	40.7
Hudson et al. (1985)	31	Fibromyalgia	25.8
Katon et al. (1985)	37	Mixed sites	32.4
Kinney et al. (1992)	50	Facial	30.0

* Chronic pain was defined as pain of more than 3 months' duration

(cont'd)

Table 1 (cont'd). Major Depressive Disorder (MDD) Among Patients with Chronic Pain

Study	n	Type of Pain	% Current MDD
Kinney et al. (1993)	90	Low back	46.0
Kramliner et al. (1983)	100	Mixed sites	25.0
Krishnan, France, & Houpt (1985)	50	Low back	68.0
Krishnan, France, Pelton, et al. (1985)	71	Low back	43.7
Large (1986)	50	Mixed sites	8.0
Linder et al. (2000)	250	Unspecified	23.0
Lindsay & Wyckoff (1981)	300	Mixed sites	87.0
Love (1987)	68	Low back	25.0
Maruta et al. (1989)	100	Mixed sites	34.0
Merskey et al. (1987)	32	Mixed sites	28.1
Muse (1985)	64	Mixed sites	1.5
Owen-Salters et al. (1996)	125	Low back	69.6
Reich et al. (1983)	43	Mixed sites	23.2
Remick et al. (1983)	68	Facial	13.2
Schaffer et al. (1980)	20	Mixed sites	50.0
Smith et al. (1994)	29	Mixed sites	58.6
Turk et al. (1995)	100	Mixed sites	8.7
Turner & Romano (1984)	40	Mixed sites	30.0
Ward et al. (1992)	81	Mixed sites	56.8
Wilson et al. (2002)	150	Mixed sites	30.0
Average:			33.6 %

Second, however, the prevalence estimates vary across a wide spectrum, and range from 1.5% to 87.0%.

There are several reasons for these disparate prevalence estimates. First, methodological issues, such as the type of diagnostic criteria employed, can affect prevalence estimates. For instance, the RDC criteria are more stringent than the DSM criteria (Sullivan et al., 1992), and can thus result in lower prevalence estimates for major depression (e.g., France, Houpt, Skott, Krishnan, & Varia, 1986; Haley et al., 1985; Kathol et al., 1990). Second, some studies have assessed other depression-spectrum disorders as well as major depression [e.g., minor depression, dysthymia, adjustment disorder with depressed mood, depressive disorder not otherwise specified (NOS); Turk, Okifuji, & Scharff, 1995]; however, prevalence estimates of other depression-spectrum disorders are not included in Table 1. The inclusion of other depression-spectrum disorders can be associated with lower rates of diagnosis of major depression if researchers tend to favour the use of less severe diagnostic categories when working with medical, as opposed to psychiatric, populations (Wilson, Chochinov, de Faye, & Breitbart, 2000). Third, the operational definition for what constitutes adequate symptom severity thresholds for the diagnosis of major depression, particularly among medical samples, can be controversial (Chochinov, Wilson, Enns, & Lander, 1994). Finally, estimates may vary considerably across studies because the DSM-IV recommends that somatic symptoms not be included in the diagnosis of depression if they are attributable to the direct physiological effects of a medical condition, thus allowing for substantial variability in clinical judgements (Banks & Kerns, 1996). Including symptoms commonly attributed to both chronic pain and depression (e.g., reduced concentration, insomnia,

fatigue) could potentially be associated with increased estimates of major depression because of a methodological confound rather than because of a clinical reality (Wilson, Mikail, D'Eon, & Minns, 2001). However, recent evidence suggests that somatic symptoms do retain their validity among samples of pain clinic patients, and they should generally be included in the diagnosis of major depression with this population (Wilson, Mikail, et al., 2001).

Research on the correlates of major depression among chronic pain patients has shown it to be associated with a variety of clinical sequelae, such as increased pain severity ratings (Benjamin et al., 1988; Currie & Wang, 2004; Elliott et al., 2003; Haley et al., 1985; Haythornthwaite, Sieber, et al., 1991), increased pain behaviours, and higher ratings of perceived life interference (Haythornthwaite, Sieber, et al., 1991). Further, the diagnosis of major depression has been associated with greater self-reported disability (Currie & Wang, 2004; Elliott et al., 2003), and increased levels of pain-related cognitive distortions (e.g., catastrophizing, overgeneralization, personalization, selective abstraction; Geisser, Roth, Theisen, Robinson, & Riley, 2000; Smith, O'Keeffe, & Christensen, 1994) among chronic pain patients.

Explaining the chronic pain - depression relationship. Three primary schools of thought have arisen in order to explain the relationship of depression to chronic pain. Some researchers have argued that pain and depression frequently co-occur because they share common biological pathways. For example, serotonin and norepinephrine are neurotransmitters that are implicated in both pain modulation and mood (Delgado, 2004; Robinson & Riley, 1999b), and some pain patients have been shown to respond favourably to antidepressant treatment (Delgado, 2004, 2006; Max, 1995), particularly to

the newer selective serotonin and norepinephrine reuptake inhibitors (e.g., SNRI's; e.g., venlafaxine, duloxetine; Delgado, 2006). Furthermore, chronic pain and depressed patients may share other physiological markers, such as increased plasma cortisol, an abnormal dexamethasone suppression test, and low levels of cerebrospinal 5-hydroxyidoleacetic acid (Diener, Van Schayck, & Kastrup, 1995; Ward et al., 1982). However, evidence for these explanations has been mixed (Romano & Turner, 1985). For instance, not all researchers have found that an abnormal dexamethasone suppression test to be characteristic of all chronic pain patients with depression (Ward, 1990). Moreover, although early studies (e.g., Pilowsky, Hallett, Bassett, Thomas, & Penhall, 1982) have not shown antidepressant treatment to be effective for chronic pain, research on the more recent dual-action antidepressants (e.g., SNRI's) and their effects on attenuating pain is still in its infancy (Delgado, 2006).

A second group of investigators has conceptualized depression as the primary disorder, with chronic pain viewed as a collection of secondary psychosomatic or neurovegetative symptoms occurring among certain susceptible or 'pain-prone' individuals (e.g., Blumer & Heilbronn, 1982). This 'masked depression' interpretation has been hotly contested. In fact, Turk and Salovey (1984) critically evaluated Blumer and Heilbronn's (1982) thesis and noted that the bulk of the empirical evidence for chronic pain as a form of masked depression was mixed, at best; they also noted that although depression is common among chronic pain patients, it is not observed universally.

Third, others have conceptualized chronic pain as the primary disorder, with depression arising secondarily as an understandable effect of persistent pain and ongoing

disability, particularly among individuals with certain vulnerabilities (Banks & Kerns, 1996; McBeth, Macfarlane, & Silman, 2002; Rudy, Kerns, & Turk, 1988). Fishbain and colleagues conducted a thorough review of the evidence in support of the above theories and found that the preponderance of evidence does indeed support this explanation (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997).

The cognitive-behavioural mediation model of depression in chronic pain can be described as a variant of the latter group of theories (Fishbain et al., 1997), as well as a diathesis-stress model (Banks & Kerns, 1996; Pincus & Williams, 1999). According to models of this kind, depression results when diatheses, or pre-existing psychological or physical vulnerability factors, interact with environmental stressors (Banks & Kerns, 1996). Borrowing heavily from the cognitive-behavioural perspectives on chronic pain (e.g., Turk & Rudy, 1986) and depression (Beck, 1976), the cognitive-behavioural mediation model proposes that cognitive appraisals and behavioural factors are the key mediators in the pain-depression relationship. In behavioural terms, chronic pain patients experience declines in instrumental and enjoyable activities and social rewards. In cognitive terms, patients' perceptions of how their pain impacts upon their lives, how they adapt to the decreased activity and decreased opportunities for social rewards associated with chronic pain, and their perceptions of control or self-efficacy, are examples of cognitive factors hypothesized to mediate the development of depression (Kerns & Haythornthwaite, 1988; Haythornthwaite, Sieber, et al., 1991; Rudy et al., 1988; Turk et al., 1995). Thus, pain *per se* is not a sufficient condition in the etiology of depression. Rather, the development of pain-related behaviours and negative cognitive appraisals are also prerequisites, inasmuch as they activate pre-existing vulnerabilities

(e.g., negative processing biases, problematic affect regulation; Kerns, 1996; Kerns & Haythornthwaite, 1988; Haythornthwaite, Sieber, et al., 1991; McBeth et al., 2002; Rudy et al., 1988; Pincus & Williams, 1999; Turk et al., 1995).

Insomnia

Like chronic pain, insomnia is a costly problem in terms of its high prevalence, its impact on reduced role function, and its association with increased use of health care services. Walsh and Englehardt (1999) estimated that the direct costs associated with insomnia (including health care services and pharmacological therapies) in the United States in 1995 totaled almost \$14 billion dollars. In a study of primary care practice attenders, Simon and Von Korff (1997) found that combined in- and out patient costs were 60% higher for patients with insomnia when compared to noninsomniac patients. Indirect costs are also considerable, because insomniacs have been shown to miss up to ten times as many work days as individuals without insomnia (Walsh, 2004; Zammit, Weiner, Damato, Sillup, & McMillan, 1999).

Insomnia in the general population. Part of the high cost associated with insomnia can be attributed to the sheer magnitude of the problem. Studies of the general population have used a wide range of definitions of insomnia, ranging from very general definitions (e.g., occasionally experiencing disturbed sleep) to the use of specific criterion-based diagnostic categories (see Table 2).

Not surprisingly, studies that used broader definitions of insomnia have yielded higher prevalence estimates than those using more strict diagnostic criteria. Mellinger, Balter, and Uhlenhuth (1985) surveyed a nationally representative sample of 3,161 American adults and found that 35% had experienced some difficulty initiating or

Table 2. DSM-IV (APA, 1994) Diagnostic Criteria for Primary Insomnia

- A complaint of difficulty initiating or maintaining sleep, or of nonrestorative sleep, for a period of at least one month.
- The sleep complaint and any associated daytime fatigue cause significant impairment in social, occupational, or other areas of functioning.
- The sleep complaint does not occur exclusively in conjunction with Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, or a Parasomnia.
- The sleep complaint does not occur exclusively in conjunction with another mental health disorder, such as Major Depressive Disorder.
- The sleep disturbance is not due to the direct physiological effects of a substance, or to a general medical condition.

maintaining sleep in the past year, while an additional 12% had experienced some form of sleep disturbance more than one year previously. Approximately half of those who had experienced some form of insomnia within the past year (17%) considered this problem to be serious. Ancoli-Isreal and Roth (1999) conducted telephone interviews with 1,000 Americans and found that 27% experienced occasional difficulty sleeping (defined as occasional insomnia, such as under conditions of heavy stress), although only 9% reported a chronic sleep disturbance, defined as difficulty sleeping on a frequent basis. Foley et al. (1999) surveyed 6,899 individuals aged 65 or older and found that 28% reported one or more symptoms of insomnia.

One of the early estimates of the prevalence of insomnia based on formal diagnostic criteria came from the Epidemiological Catchment Area Study in the United States (Ford & Kamerow, 1989). Ford and Kamerow found that 10.2% of their sample of 7,954 community participants reported insomnia that fulfilled the diagnostic criteria of the DSM-III. Other studies of the general population have generally confirmed the high prevalence of insomnia. Breslau, Roth, Rosenthal, and Andreski (1996) surveyed 1,007 21- to 30 year-old members of a United States health maintenance organization and found that a diagnosis of insomnia had been made in 16.6% of cases. In a sample of 2,370 adults in the community aged 50 and older, Roberts et al. (2000) found that 23% reported trouble falling or staying asleep nearly every day for the previous two weeks.

Research conducted in European countries has been consistent with these estimates. Ohayon (1997) reported the results of a telephone survey of 5,622 respondents in France, and found that 18.6% reported one or more insomnia complaints (e.g., difficulty initiating or maintaining sleep, or both) at the time of the interview. In addition, 15.3% of the total

sample complained of one or more symptoms for at least one month, and 12.7% of the total sample acknowledged negative daytime repercussions, thus satisfying DSM-IV criteria. Another telephone survey of 4,972 community subjects in the United Kingdom yielded similar results. In that study, Ohayon et al. (1997) reported that 36.2% of their sample experienced at least one insomnia-related symptom at the time of the interview, while 8.7% of the sample complained of one or more symptoms of insomnia in addition to being dissatisfied with their sleep. These findings were integrated into a larger study of 14,915 participants recruited by telephone from the general populations of the UK, Germany, Italy, and Portugal. In this study (Ohyaon & Roth, 2003), 19.1% of participants reported at least one symptom of insomnia accompanied by negative daytime consequences, and over 90% of these individuals reported that their insomnia had lasted for six months or more. In another large-scale study, Chevalier et al. (1999) surveyed members of the general population within five European countries (Belgium, Germany, Great Britain, Ireland, and Sweden). Rates of severe insomnia (defined as the presence of two or more sleep complaints on three or more nights per week with sleep-related sequelae) ranged from a low of 4% in Germany to a high of 22% in Great Britain.

More recently, Morin, LeBlanc, Daley, Gregoire, & Merette (2006) conducted a telephone survey of 2,001 randomly selected French-speaking adults in the province of Quebec. Of the respondents, 29.9 % reported one or more symptoms of insomnia (e.g., difficulty initiating or maintaining sleep, or early morning awakening) three or more nights per week, although only 16.9 % of the total sample expressed dissatisfaction with their sleep. Further, 9.5% of the sample reported symptoms consistent with the diagnostic criteria of insomnia. Thus, occasional insomnia affects a significant portion of the general

population, and between 4% and 23% of survey respondents appear to have a more chronic problem characteristic of a discrete sleep disorder as defined in the DSM-IV.

Correlates of insomnia in the general population. A variety of demographic variables have been associated with insomnia in the general population; these include female gender (Bixler, Vgontzas, Lin, Vela-Bueno, & Kales, 2002; Chevalier et al., 1999; Ford & Kamerow, 1989; Hohagen et al., 1994; Kuppermann et al., 1995; Mellinger et al., 1985; Morin et al., 2006; Ohayon, 1997; Ohayon et al., 1997, 1998; Ohayon & Roth, 2003; Roberts, Shema, Kaplan, & Strawbridge, 2000), increased age (Ancoli-Isreal & Roth, 1999; Ford & Kamerow, 1989; Mellinger et al., 1985; Morin et al., 2006; Ohayon, 1997; Ohayon et al., 1997, 1998; Ohayon & Roth, 2003; Roberts et al., 2000), lower socio-economic status (Roberts et al., 2000), lower level of education (Ancoli-Isreal & Roth, 1999; Ford & Kamerow, 1989; Roberts et al., 2000), and being separated or widowed (Ford & Kamerow, 1989).

Studies on the correlates of insomnia in the general population have also shown that poor sleep is associated with self-reports of diminished cognitive function (Moul et al., 2002), low mood (Morin et al., 2006; Ohayon & Roth, 2001), poor health (Bixler et al., 2002; Hasler et al., 2005; Morin et al., 2006), and negative social repercussions (Hasler et al., 2005). Kuppermann et al. (1995), for example, found that self-described insomniacs scored significantly worse than noninsomniacs on measures of psychological distress, physical health, energy, cognitive functioning, work satisfaction, and job performance. Roth and Ancoli-Israel (1999) found that insomniacs were significantly less likely to rate as 'excellent' or 'good' their physical health, memory, ability to concentrate, accomplish tasks, and handle irritations. Zammit et al. (1999) surveyed 261 community adults and

found that insomniacs reported worse general health, and significantly higher levels of depression and anxiety than individuals without insomnia complaints. These results are further underscored by the fact that Zammit et al. (1999) had excluded participants with a history of diagnosed psychiatric disorders. Consistent with other investigators, they also found that insomniacs generally reported poorer attention, concentration, memory, reasoning, problem solving, and reaction times when compared to noninsomniacs. Alapin et al. (2000) studied older community adults, and also a group of university students and found that poor sleepers reported more negative daytime repercussions (fatigue, sleepiness, diminished concentration) than good sleepers. Insomnia has also been associated with decreased quality of life ratings (Hasler et al., 2005; Leger, Scheuermaier, Philip, Paillard, & Guilleminault, 2001; Zammit et al., 1999), and lower self-reported enjoyment of interpersonal relationships (Roth & Ancoli-Israel, 1999).

Other findings indicate that the experience of pain is another important correlate of insomnia. For example, both Kuppermann et al. (1995) and Zammit et al. (1999) found that insomniacs reported higher levels of physical pain relative to noninsomniacs. Thus, the literature on the correlates of insomnia in the general population indicates that insomnia is associated with multiple negative health, cognitive, and psychological sequelae, including the experience of pain.

Insomnia in primary care. Estimates of the prevalence of insomnia among attenders of primary care clinics are generally higher than those observed in the general population. Shochat et al. (1999) surveyed 286 patients from American primary care clinics and found that 50% reported occasional insomnia (insomnia occurring only at certain times, such as under heavy stress), while an additional 19% reported chronic insomnia. Hohagen

et al. (1993) surveyed 2,512 German general practice patients and found that 18.7 % satisfied DSM-III-R diagnostic criteria for insomnia, while 12.2% experienced moderate levels of insomnia (defined as difficulties initiating or maintaining sleep but without impaired daytime functioning). A further 15% experienced mild insomnia (defined as occasional difficulties initiating or maintaining sleep). In a subsequent study by these researchers (Hohagen et al., 1994), 330 German general practice attenders aged 65 and older were assessed using DSM-III-R criteria. They found that 23% experienced severe insomnia, while an additional third reported moderate (17%) and mild (17%) sleep problems. Finally, Simon and Von Korff (1997) surveyed 1,962 members of an American health-maintenance organization. Using a very strict definition (i.e., taking two or more hours to fall asleep at night, for a period of two or more weeks), they found that 10% of the sample met criteria for severe sleep-onset insomnia. However, this definition would not have identified patients with sleep maintenance complaints, which is actually the more common sleep disturbance among the medically ill (Rodin, McAvay, & Timko, 1988).

Correlates of insomnia in primary care. Consistent with the literature on insomnia in the general population, insomnia among primary care patients also appears to have wide-reaching implications. Shochat et al. (1999) found that patients with chronic insomnia reported significantly poorer daytime functioning when compared to those with occasional insomnia. Specifically, chronic insomniacs were found to score significantly lower on self-reported measures of physical health, cognitive function, interpersonal relationships, quality of life, and mood. Hohagen et al. (1993) also recorded a significant association between disturbed sleep and complaints about impaired health status.

Depression is an important correlate of insomnia in primary care. Hohagen et al. (1993) found that severe insomnia was associated with all psychiatric diagnoses, while moderate insomnia was only associated with the diagnosis of depression (assessed via unstructured clinical interview). In a subsequent study, insomnia was found to be correlated significantly with self-reported depressive symptoms (Hohagen et al., 1994). Schramm, Hohagen, Kappler, Grasshoff, and Berger (1995) examined 105 self-reported disturbed sleepers and found that 63% met DSM-III-R criteria for current insomnia. Of these, 24% had a concurrent diagnosis of a mood disorder. Simon and Von Korff (1997) reported that primary care patients with current insomnia were significantly more likely to suffer from a current depressive disorder than patients without current insomnia. Insomnia was also associated with significantly greater self- and interviewer-rated levels of disability. Furthermore, insomnia appeared to exert a greater impact upon individuals who also had concurrent depression. These findings are not surprising, however, given the frequent comorbidity of depression and insomnia; this will be discussed in greater detail in a subsequent section.

The Treatment of Insomnia

The pharmacological treatment of insomnia has traditionally been the intervention of choice within the medical community (Bliwise, 1991). However, for a variety of reasons, sedative-hypnotics are not recommended for long-term use (United States' National Institutes of Health, 1988, 1991). They have been associated with poor quality sleep (e.g., Kales & Kales, 1987), adverse daytime functioning (Johnson & Chernik, 1982), the development of tolerance and dependence (Espie, 1991), and rebound insomnia following abrupt cessation (Moran & Stoudemire, 1992). Newer nonbenzodiazepine hypnotics,

such as zolpidem and zalepon appear to possess lower abuse potential than traditional sedative-hypnotics, but additional studies are necessary to evaluate their therapeutic safety and efficacy for long term use (see Thase, 2005, for a recent review). Given these problems, there has been considerable interest in developing nonpharmacological treatments for insomnia. Currently, the most dominant approach follows a cognitive-behavioural model.

Cognitive-Behavioural Theory of Insomnia

Current conceptualizations of insomnia view it as a complex, multifactorial process that can result from a variety of biopsychosocial influences rather than any single cause. According to Morin (1993), cognitive and behavioural factors interact with physiological and affective factors to bring about disturbed sleep. A model of chronic insomnia is shown in Figure 1. In this paradigm, primary psychophysiological insomnia is thought to develop after an acute, precipitating life event that initially results in transiently disturbed sleep, caused mainly by stress or worry. In support of this contention, about 75% of insomniacs report that their insomnia developed at about the same time as one or more stressful life events (Gagne, Bastien, & Morin, 1997; Healey et al., 1981). If adaptation to the initial stressor and resumption of normal sleep does not occur, a chronic sleep disorder may develop among predisposed individuals. People may be predisposed to insomnia on the basis of presumed genetic influences on the depth of sleep, or because of other physiological reasons (Morin, 1993; Smith & Perlis, 2006; Spielman, Caruso, & Glovinsky, 1987). For example, Kales and Kales (1984) found that insomniacs are more prone to nocturnal and diurnal physiological arousal than other sleepers. From a psychological perspective, people may also be predisposed to develop

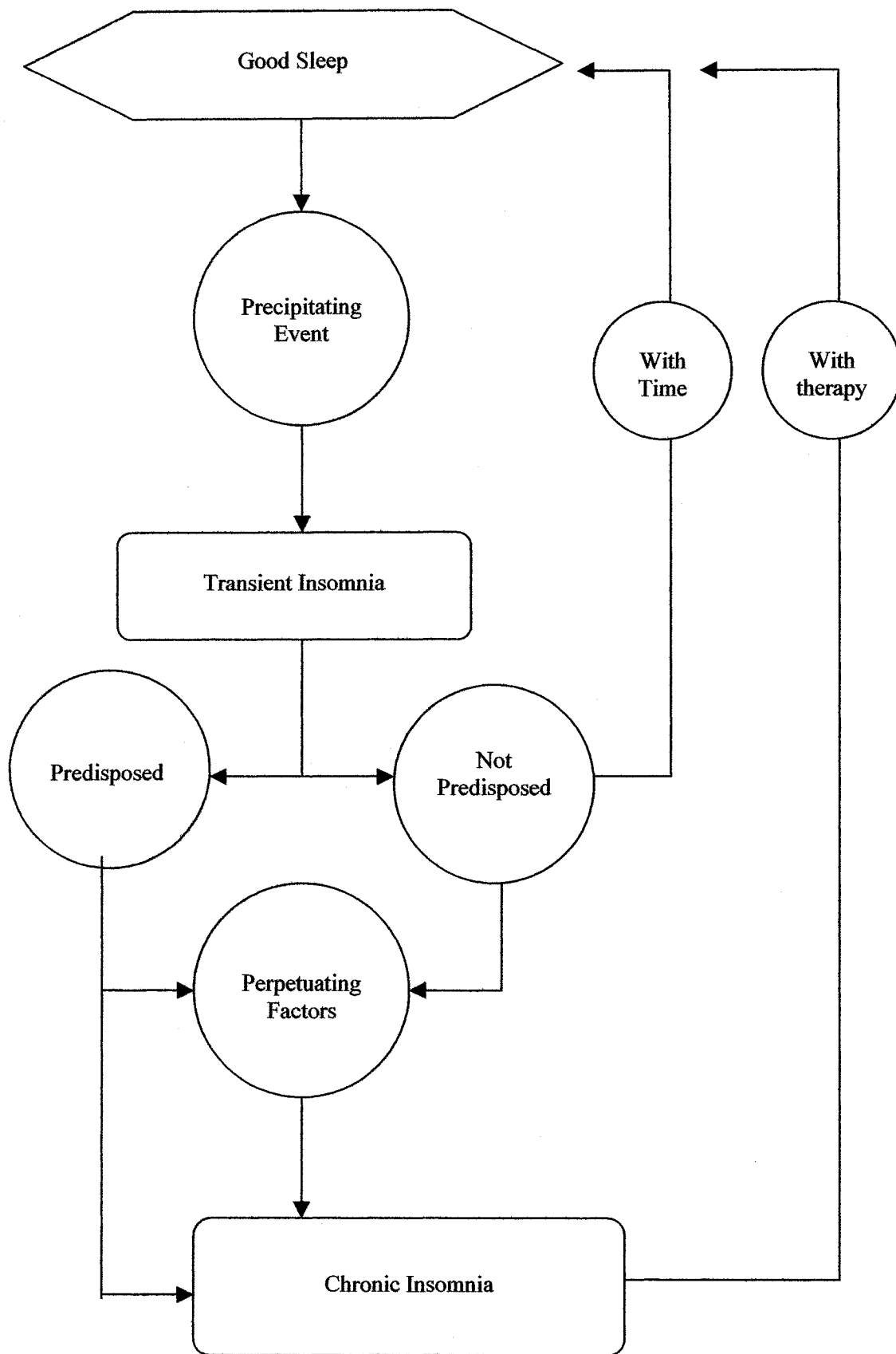


Figure 1. The Development Of Chronic Insomnia. Adapted from Spielman et al. (1996).

insomnia if they are prone to reacting to stress with mental rumination, negative 'racing' thoughts, or more generalized feelings of anxiety.

The ensuing sleep disturbance is perpetuated and exacerbated by multiple factors that arise in an attempt to cope with the initial period of sleeplessness. As can be seen in Figure 2, these factors are cognitive and behavioural in nature and can be conceptualized as falling within one of two general classes of phenomena, related to either conditioning or coping processes. In terms of conditioning, chronic insomnia develops when, during the initial period of sleeplessness, the bed and the pre-sleep context become associated, through classical conditioning, with wakefulness and arousal. Rather than remaining strong conditional stimuli that promote sleep onset, these bedroom cues become conditional stimuli that trigger a state of cognitive hyperarousal which is characteristic of chronic insomnia. This state of hyperarousal, with its attendant rumination (e.g., excessive worrying about a sleep problem and its impact on one's life), is particularly incompatible with restful sleep (Morin, 1993; Smith & Perlis, 2006; Spielman et al., 1987).

In addition, chronic insomniacs adopt a variety of coping strategies in an attempt to deal with the initial sleep loss, such as napping during the day, spending excessive amounts of time in bed, and over-reliance upon medications for sleep. These coping methods can be problematic in the long-term, however, and may serve to perpetuate the sleep disturbance (Morin, 1993; Smith & Perlis, 2006; Spielman et al., 1987). Given the presumed importance of psychological factors in the etiology and maintenance of this pervasive sleep disorder, chronic primary insomnia is sometimes referred to as 'learned,' 'conditioned,' or 'psychophysiological' insomnia.

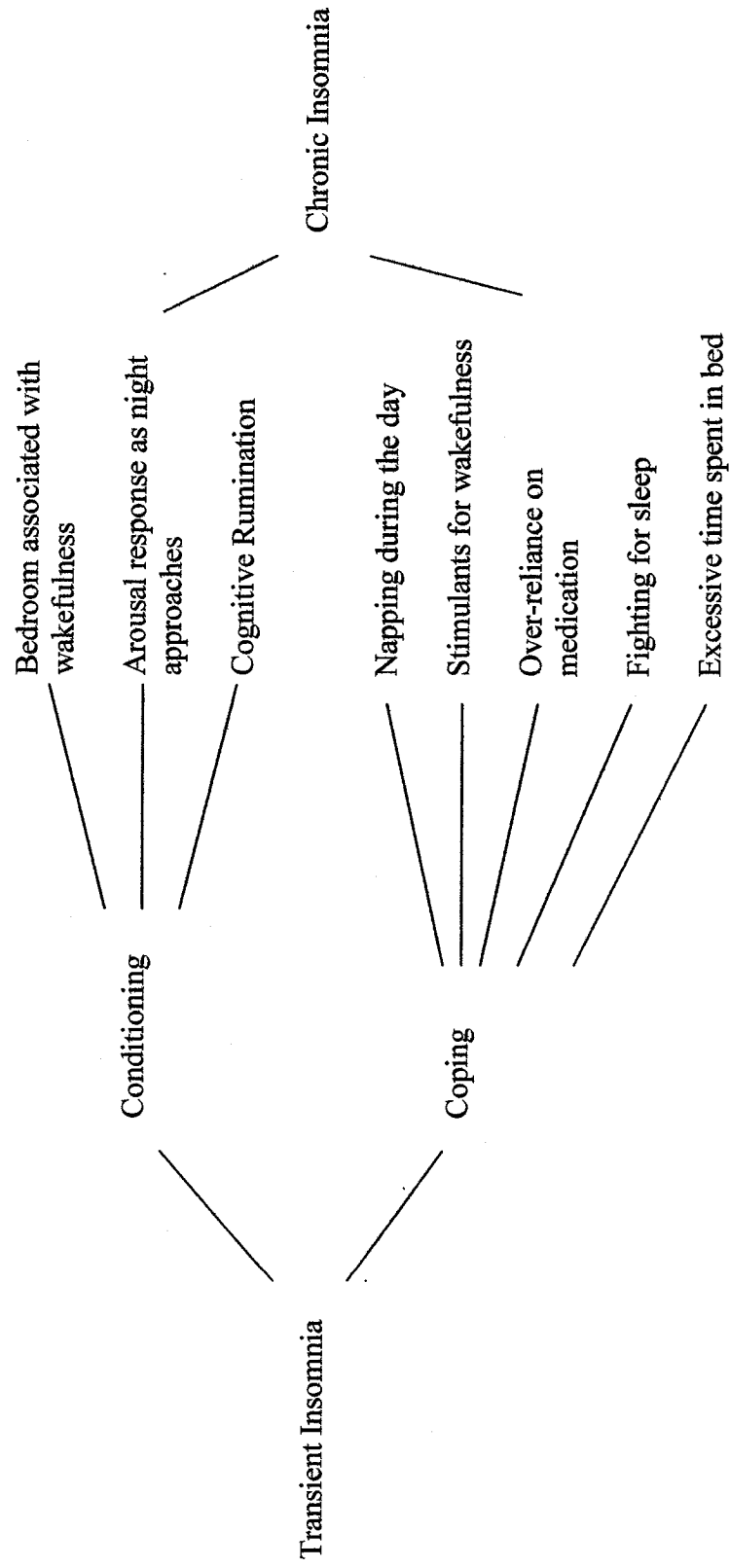


Figure 2. Perpetuating Factors of Insomnia

Cognitive-behavioural therapy for insomnia. CBT for primary insomnia is based on two traditions of psychotherapy. The first tradition is behaviour therapy, and consists of specific procedures designed to reduce cognitive and somatic arousal, eliminate sleep-incompatible behaviours, and increase the association between environmental stimuli (e.g., one's bed and bedroom) with sleep. The second tradition is cognitive therapy, which seeks to alter dysfunctional thoughts and attitudes related to sleep and the impact of sleep loss on daily life (Morin, 1993).

In behavioural terms, sleep restriction (Spielman et al., 1987) and stimulus control (Bootzin, Epstein, & Wood, 1991; Spielman et al., 1987) therapy are commonly instituted in order to decrease the sleep-incompatible behaviours developed during the period of initial sleeplessness. In the case of sleep restriction, patients are instructed to remain in bed only for the average amount of time they had reported sleeping in the week previously. The period of time allowed in bed is then increased until the patient achieves an acceptable level of sleep efficiency (total sleep time/total time in bed), usually defined as 85% or more (i.e., the patient is asleep for at least 85% of the time spent in bed). In the case of stimulus control, patients are instructed to use the bed and bedroom only for sleep and sexual activities, and are instructed to leave the bedroom if unable to sleep within 15-20 minutes of lying down (Morin, 1993; Morin et al., 1993). Because it is assumed that, over time, the pre-sleep environment has come to be associated with arousal and sleeplessness instead of rest and relaxation, these therapies are instituted to increase levels of sleepiness at bedtime and to strengthen the association of bedroom stimuli with sleep (Spielman et al., 1987).

Relaxation therapy and sleep hygiene education are other common behavioural interventions (Smith & Perlis, 2006). Relaxation therapy seeks to decrease somatic and/or cognitive anxiety (Morin et al., 1999; Smith & Perlis, 2006). To counteract cognitive anxiety or rumination, techniques such as imagery training and thought stopping may be incorporated into a relaxation regimen. Sleep hygiene education is a common psychoeducational component of CBT for insomnia. This intervention seeks to educate patients about sleep-incompatible habits such as the effects of caffeine, alcohol, tobacco, nicotine, and sedatives on sleep (Morin, 1993).

According to the cognitive-behavioural model of insomnia, insomniacs tend to engage in maladaptive cognitive strategies that serve to perpetuate the sleep problem. The cognitive therapy component of CBT therefore seeks to address and alter these dysfunctional thoughts through the use of cognitive restructuring procedures. Insomniacs often have unrealistic sleep expectations, make misattributions about the causes of insomnia, and hold inaccurate beliefs regarding the negative consequences of sleeplessness. For instance, insomniacs often hold the false belief that they must have eight hours' sleep each night, or risk serious adverse effects on health and productivity (Morin, 1993; Smith & Perlis, 2006).

Insomniacs also engage in a cognitive style characterized by excessive rumination about the sleep problem, magnification of its consequences, and catastrophizing about its impact. It is unclear as to whether or not this state of cognitive hyperarousal is the cause or consequence of nighttime wakefulness (Morin, 1993). Nevertheless, it can contribute greatly to the perpetuation of the vicious cycle of sleeplessness, increased worry, and the

likelihood of increasing chronicity for the insomnia; therefore it is also a target of intervention (Morin, 1993).

It is beyond the scope of the present review to conduct a thorough analysis of the efficacy of CBT for insomnia. It should be noted, however, that the available literature has been the subject of two meta-analyses (Morin et al., 1994; Murtagh & Greenwood, 1995), a United States' National Institutes of Health (NIH) Technology Assessment Review (1996), and a task force report from the American Academy of Sleep Medicine (Chesson et al., 1999; Morin et al., 1999).

In general, it appears that 70%-80% of patients benefit from nonpharmacological treatment for chronic insomnia (Morin et al., 1999). The typical patient can expect an average increase of approximately 30 minutes of total sleep time (Espie, Inglis, & Harvey, 2001), while sleep onset latency and/or time awake after sleep onset are also reduced substantially. Patients can also expect that their subjective sleep quality and satisfaction with sleep patterns will improve. Furthermore, treatment gains have been shown to be maintained for six months to three years post treatment (Backhaus, Hohagen, Voderholzer, & Riemann, 2001; Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Espie et al., 2001; Morin, Blais, & Savard, 2002).

Chronic Pain and Insomnia

The fact that chronic pain is associated with insomnia is well established in the literature. Estimates of disturbed sleep among pain clinic patients have shown that substantial numbers are affected by this problem. These studies are summarized in Table 3. As is evident, the prevalence of disturbed sleep in samples of pain clinic attenders are among the highest ever reported for any specific population, with estimates ranging from

Table 3. Prevalence Estimates of Disturbed Sleep Among Patients with Chronic Pain

Study	n	Type of Pain	% and Type of Insomnia
Atkinson et al. (1988)	51	Low back	51% 'moderately' or 'very' unsatisfactory sleep
Becker et al. (1997)	150	Mixed sites	42% 'poor' sleepers
McCracken & Iverson (2002)	287	Mixed sites	88.9% one or more insomnia complaints
Morin et al. (1998)	105	Mixed sites	65% 'poor' sleepers
Nicassio & Wallston (1992)	242	RA*	57% 'restless' sleep, most of the time
Pilowsky et al. (1985)	100	Mixed sites	78% 'poor' sleepers
Sayar et al. (2002)	40	Mixed sites	57.5% 'poor' sleepers
Smith et al. (2000)	51	Mixed sites	88% one or more insomnia Complaints
Wilson et al. (2002)	50	Mixed sites	64% clinically significant insomnia

* Rheumatoid Arthritis

42% to 88.9% of patients. It is noteworthy, however, that none of these studies has applied a formal, DSM-IV diagnostic approach to the assessment of insomnia in patients with chronic pain.

Nevertheless, not only is insomnia highly prevalent among individuals with chronic pain, it also appears to be quite severe. For example, Wilson, Eriksson, D'Eon, Mikail, and Emery (2002) had psychologists make clinical global ratings for a group of 150 pain clinic attenders. Of the 64% of patients who were considered to have a significant sleep problem, 11.3% were rated as having only mild insomnia, 12.7% had a moderate problem, 34% had severe insomnia, and 6% had a sleep disturbance that was rated as extremely severe.

Correlates of Insomnia in Chronic Pain.

Insomnia among chronic pain patients has been associated with a variety of adverse pain- and mood-related phenomena. Studies of this kind typically use one of the following research methodologies: (1) comparisons of good and poor sleepers, (2) within-group correlational investigations, (3) polysomnography, and (4) ambulatory monitoring of nocturnal movement.

Studies comparing good and poor sleepers. One group of studies examining the impact of insomnia upon individuals with chronic pain has compared groups of self-reported good and poor sleepers (Atkinson et al., 1988; Morin et al., 1998; Pilowsky et al., 1985). These studies have found that self-described 'poor' sleepers sleep fewer hours per night than self-described 'good' sleepers. Furthermore, Morin et al. (1998) found that poor sleepers took longer to fall asleep, experienced more nocturnal awakenings, and reported increased levels of daytime sleepiness.

These studies have also shown that insomnia has implications that extend beyond the realm of sleep and into the experience of pain and emotional well being. For example, poor sleepers have reported higher levels of pain severity (Atkinson et al., 1988; McCracken & Iverson, 2002; Morin et al., 1998; Pilowsky et al., 1985; Sayar, Arikan, & Yontem, 2002), in addition to elevated levels of depressive symptoms (Atkinson et al., 1988; Pilowsky et al., 1985). Although the latter finding was not replicated by Morin et al. (1998), these authors questioned the sensitivity of their single, visual-analogue measure of depression, and they did find that poor sleepers reported more 'pain unpleasantness', a measure of the affective response to pain. Wilson et al. (2002) found that patients with insomnia had higher scores on the McGill Pain Questionnaire (indicating a more severe pain experience) than patients without insomnia. Further, insomnia severity was found to be a significant predictor of pain severity in regression analyses, even after depression was held constant. Patients with insomnia also had elevated levels of affective distress relative to the comparison group, even when those who were diagnosed with major depression were excluded.

Within-group correlational studies. Other researchers have examined the correlates of insomnia among chronic pain patients using within-group, correlational analyses. Several of these studies have been based on survey designs (Goodrich, Cogan, Randolph, & Racz, 1998; Nicassio, Moxham, Schuman, & Gevirtz, 2002; Ohayon, 2005; Smith, Perlis, Smith, Giles, & Carmody, 2000; Menefee et al., 2000), whereas other researchers have used prospective self-monitoring via sleep diaries (Affleck et al., 1996; Haythornthwaite, Hegel, & Kerns, 1991).

A number of surveys have shown sleep disturbance to be associated with increased pain severity and disability (McCracken & Iverson, 2002; Nicassio et al., 2002; Sayar et al, 2002). Ohayon (2005) reported that insomnia with comorbid chronic pain is associated with significantly poorer daytime functioning than insomnia without comorbid pain. However, the data also suggest that insomnia among chronic pain patients has implications that extend beyond sleep and into the experience of pain and mental health (McCracken & Iverson, 2002; Sayar et al., 2002). Smith and colleagues found that chronic pain patients displayed profiles on the Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) that were similar to the published norms for primary insomniacs, with the exception that pain patients tended to report that they had even greater difficulty falling asleep and a higher frequency of nocturnal awakenings. Furthermore, pain severity and depressive symptomatology were significantly associated with poor self-reported sleep quality. However, in regression analyses, pain severity was not a significant predictor of self-reported sleep quality, although presleep cognitive arousal and, to a lesser extent, general activity level, were significant predictor variables (Smith et al., 2000; Smith, Perlis, Carmody, Smith, & Giles, 2001).

Goodrich et al. (1998) found that indices of disturbed sleep (poor sleep quality, increased sleep onset latency, and decreased sleep quantity) were significant predictors of pain intensity and pain frequency in regression analyses. However, these variables only accounted for approximately 16% and 13% of the variance in pain intensity and pain frequency scores, respectively. The addition of self-reported depressive, anxiety, and obsessive-compulsive scores added only about 2% to the explained variance in pain intensity; further, when these personality variables were regressed on pain frequency, the

equation was rendered nonsignificant. None of the personality variables was significantly correlated with pain intensity, although they were significantly correlated with increased sleep onset latency and decreased sleep quality. The fact that sleep variables were better predictors of pain than indices of psychological distress highlights the importance of explicitly addressing sleep disturbance in patients with chronic pain (Goodrich et al., 1998).

Menefee et al. (2000) found that lower ratings of physical functioning and longer duration of pain were associated with increased sleep onset latency and poor sleep quality. Further, ratings of highest pain within the past two weeks served as significant, individual predictors of poor sleep quality and increased sleep onset latency. Depressed mood was associated with increased daytime sleepiness, although pain was not. These findings suggest that degree of physical functioning and pain are more important determinants of disturbed sleep than depressed mood, and the authors suggest that degree of physical functioning be considered in future research (Menefee et al., 2000).

Taken together, these studies suggest that insomnia and pain are interrelated, but the findings regarding depression and other psychosocial variables are not as straightforward. More research is clearly necessary in order to disentangle the relative influences of factors such as depression and disability on insomnia and pain.

Prospective self-monitoring studies are advantageous in that this method reduces recall bias that may affect global, retrospective estimates of sleep parameters. Another advantage is that prospective self-monitoring allows for the tracking of daily fluctuations in sleep, pain, and mood. Haythornthwaite, Hegel, et al. (1991) studied 46 mixed-site chronic pain patients over four days and found that total sleep time was correlated

negatively with pain intensity; daytime pain intensity was in turn correlated with difficulties initiating sleep the following night. However, nocturnal and early-morning awakenings were not correlated with pain intensity. Haythornthwaite, Hegel, et al. (1991) thus concluded that pain and insomnia may exert bidirectional influences on one another: increased pain may contribute to insomnia and insomnia may increase pain intensity. The authors also found that depression and anxiety were associated with fewer hours' sleep and poor sleep quality. In fact, these correlations were significantly higher than those observed between sleep parameters and pain intensity, suggesting that mental health problems, rather than pain *per se*, may play a greater role in insomnia.

Affleck et al. (1996) studied 50 fibromyalgia patients over 30 consecutive days. They found that sleep quality and pain intensity were inversely correlated, and that this relationship was largely mediated by the amount of attention paid to pain. Not surprisingly, more attention to pain was reported on days when pain was more intense. Greater attention to pain was then associated with poorer sleep, which in turn perpetuated the cycle of increased pain and attention to pain. Affleck et al. (1996) thus concluded that cognitive and emotional orientation toward pain during the day (which would be more likely as pain increases) is particularly incompatible with good sleep. Insomnia, then, may amplify the pain experience on subsequent days.

Nicassio et al. (2002) conducted one-week longitudinal analyses, in addition to cross-sectional analyses mentioned above, within a sample of 63 FM patients. They found that daytime pain was a significant predictor of poor sleep quality that night, which, in turn, was associated with increased fatigue the next day. Thus, studies of this kind, like those comparing good and poor sleepers, also show meaningful associations

among pain, sleep, and mood. Moreover, they provide evidence for reciprocal influences across the three domains.

Polysomnographic studies. The studies described to this point have relied upon subjective, self-report measures. Although clinically important, these methods are arguably vulnerable to recall bias and the inaccurate reporting of specific sleep parameters (Mayers & Baldwin, 2006). For this reason, objective methods, such as polysomnography and actigraphy, have also been used in assessments of insomnia.

In general, the disturbed sleep of chronic pain patients can be characterized as mirroring that of psychophysiological insomnia, in the sense that people with chronic pain report trouble initiating and maintaining sleep, poor sleep quality, and adverse daytime functioning (American Sleep Disorders Association [ASDA], 1990). In fact, Schneider-Helmert et al. (2001) reported that objective (polysomnographic) and subjective sleep profiles of chronic pain patients did not differ significantly from those of primary psychophysiological insomniacs (e.g., both groups showed low total sleep time and poor sleep efficiency).

Polysomnography (PSG) is a multi-modal assessment method that objectively records several physiological indices of sleep, such as electrical activity in the electroencephalograph (thus permitting the identification of distinct sleep stages), respiration, and muscular tension. In the first PSG study of chronic pain patients, Wittig et al. (1982) compared their PSG profiles with those of psychiatric patients, and 'subjective' insomniacs (people whose sleep problems are believed to be subjective only, and due to a sleep-state misperception). Wittig et al. (1982) found that the average sleep efficiency (percentage of time in bed that is spent asleep) of pain patients was

significantly lower (74.6%) than that of subjective insomniacs (89.5%), and well below the 85% criterion traditionally used to differentiate good and poor sleepers (Lacks & Morin, 1992). These findings were generally confined to the sleep parameters of time awake before falling asleep and time awake during the night, rather than to early morning awakenings. However, the disturbed sleep of psychiatric patients, with a sleep efficiency of 62.4%, was largely accounted for by early morning awakenings. Unfortunately, several important details were not reported in this study, such as the diagnoses of the psychiatric participants or their medication status.

Other PSG studies have shown that chronic pain patients display a range of sleep-related anomalies. For instance, researchers have reported a specific anomaly in the EEG known as alpha-delta sleep among some patients with fibromyalgia (Carette, Oakson, Guimont, & Steriade, 1995; Moldofsky et al, 1975; Roizenblatt, Moldofsky, Benedito-Silva, & Tufik, 2001), rheumatoid arthritis (Mahowald, Mahowald, Bundlie, & Ytterberg, 1989; Molofsky et al., 1983), and chronic pain (Schneider-Helmert, Whitehouse, Kumar, & Lijzenga, 2001; Wittig et al., 1982). Alpha waves are associated with wakefulness, so their presence during sleep (especially during stage four or delta sleep, the deepest level) is thought to disrupt the restorative function of sleep (Perlis, Giles, Bootzin, et al., 1997).

Alpha activity during sleep has been correlated with self-reported fatigue and sleep difficulty (Carette et al., 1995) and poor sleep (Roizenblatt et al., 2001) among fibromyalgia patients. Roizenblatt et al. (2001) found that patients with alpha-delta sleep reported significantly more post-sleep tender points than patients whose sleep is not characterized by alpha intrusions. However, in two other studies of fibromyalgia patients, alpha activity was not significantly correlated with pain symptoms (Carette et al., 1995;

Perlis, Giles, Bootzin, et al., 1997). These discrepancies, in addition to the fact that alpha-delta sleep is not seen in all chronic pain patients with disturbed sleep, and has been observed among other patient groups, including primary insomniacs (Schneider-Helmert et al., 2001) and individuals with no apparent medical or psychiatric problems (Scheuler, Stinsoff, & Kubicki, 1983) have led some investigators to question its significance. In fact, Schneider-Helmert et al. (2001) found comparable rates of alpha sleep among primary insomniacs (72%) and pain patients (62%), thus suggesting that alpha sleep is not uniquely associated with pain syndromes. Thus, the role of alpha-delta PSG anomalies as a physiological mechanism related to insomnia secondary to chronic pain is still poorly understood.

Other physiological abnormalities have been implicated in the disturbed sleep of people with chronic pain, including reduced latency to rapid eye movement (REM) sleep and periodic leg movements. Atkinson et al. (1988) and Blumer et al. (1982) found that 40% of patients with chronic pain entered REM sleep more quickly than expected. Periodic leg movements, or 'restless legs', were recorded in three of Wittig et al.'s (1982) 26 patients, and in six of the seven patients studied by Atkinson et al. (1988). Nevertheless, these abnormalities were not observed universally, and they have been recorded among primary insomniacs as well (see Wittig et al., 1982). Thus, as is the case with alpha-delta sleep, the significance of these factors in the insomnia of chronic pain patients remains unclear.

In summary, PSG studies have confirmed the presence of a significant sleep disturbance among chronic pain patients who complain of insomnia. These studies have also elucidated the nature of this sleep disturbance, which is characterized predominantly

by delayed sleep onset and decreased sleep efficiency, as opposed to early morning awakenings (Wittig et al., 1982). These studies have also yielded conflicting evidence regarding pain and PSG anomalies, and have shown that some individuals display periodic limb movements and reduced REM latencies. Although these problems may contribute to the severity of the sleep disturbance for some patients, for the majority, the problems with initiating and maintaining sleep represent the most significant issues.

Ambulatory monitoring studies. Although PSG is the accepted 'gold standard' of sleep assessment, it nevertheless has several disadvantages. PSG is expensive, it is typically conducted in a specialized sleep laboratory, and it involves intrusive electrophysiological monitoring. This can impact adversely on participant recruitment, and an adaptation period of one or two nights is usually required in order to obtain a valid assessment. In response to this, an increasing number of studies have used ambulatory actigraphic monitoring to measure sleep parameters. With actigraphy, a relatively nonintrusive, motion-sensitive detector is worn by the participant while asleep, usually on the nondominant wrist. The rationale behind the use of actigraphy in sleep studies is that periods of wakefulness are associated with more frequent movements than are periods of sleep (Ancoli-Israel et al., 2003; Wilson et al., 1998). The concordance rate of actigraphy and PSG for distinguishing periods of wakefulness from sleep has been in the range of 78%-98% of epochs scored (see Sadeh, Hauri, Kripke, and Lavie, 1995, for a review).

To date, few studies have used actigraphy to evaluate sleep among individuals with chronic pain. Lavie et al. (1992) conducted actigraphic sleep assessments among healthy controls and small groups of patients with rheumatoid arthritis (RA) or chronic low back pain. The actigraphic data showed that patients with arthritis displayed the

lowest sleep efficiency (78%), which was significantly lower than that of controls (93%); the sleep efficiencies of back pain patients occupied an intermediary position (86%). Both patient groups displayed more awakenings than did the control participants. Lavie et al. (1992) also found that evening levels of self-reported pain were correlated significantly with actigraphically-assessed sleep efficiency, while nighttime pain levels were associated with high overall activity during the night. In general, Lavie et al. (1992) concluded that actigraphic monitoring yields valid assessments of sleep parameters in patients whose sleep is disturbed by chronic pain.

Wilson et al. (1998) studied 40 chronic pain patients for two consecutive nights via actigraphy and sleep diaries. Both methods consistently demonstrated low sleep efficiency among these patients (76% and 65% for actigraphy and sleep diaries, respectively), a relatively long period of time awake after sleep onset (110 and 83 minutes, on average), and low total sleep time (5.8 and 5.3 hours). Concordance rates between actigraphy and self-reports were significant, albeit only moderate ($r_s = .38$ to $.44$).

Finally, Korszun et al. (2002) examined the sleep of 22 fibromyalgia patients using actigraphy. They found increased nighttime activity levels among these patients relative to normal controls.

In summary, studies using actigraphic assessments of sleep have shown that this methodology is sensitive to the sleep disturbance among chronic pain patients, and confirm the clinical significance of the sleep disturbance.

Etiological Explanations of the Pain – Insomnia Relationship

Although there is little disagreement over the basic fact that many people with chronic pain struggle with insomnia, the reasons for the sleep disturbance are not entirely clear. In general, there are four main explanations for the pain-insomnia relationship: (1) pain causes insomnia, (2) insomnia causes pain, (3) affective distress mediates the pain-insomnia link, and (4) the cognitive-behavioural model of insomnia secondary to chronic pain.

Pain causes insomnia. The notion that there is a straightforward, linear relationship between pain nociception and insomnia is intuitively appealing, particularly in light of the research regarding patients' own opinions regarding their insomnia. Chronic pain patients tend to attribute their poor sleep directly to their pain, say that their pain is sufficiently discomforting and distracting to prevent them from initiating and maintaining sleep, and that the sleep that they do get is not restorative (Morin et al., 1998; Nicassio & Wallston, 1992; Wilson, Mikail, et al., 2001). For example, Smith et al. (2000) found that 60% of their sample of cited pain as the 'sole' reason for their insomnia. In addition, in a two-year longitudinal study of RA patients, Nicassio and Wallston (1992) found that pain at time 1 could predict the development of sleep disturbance at time 2, whereas the inverse was not found to be the case. Therefore, pain does seem to be an initiating experience for insomnia for many individuals, and patients themselves tend to attribute their poor sleep to pain.

Although insomnia is common among chronic pain patients, it is not a universal phenomenon. In fact, all of the relevant research has found that some chronic pain patients do indeed sleep well. This may be related to a normative process of nociceptive

attenuation during sleep. Lavigne et al. (2000) administered thermal stimulation to healthy volunteers and found that various objective measures of sleep quality (sleep stage shift in the EEG, awakening) were not affected by nociceptive stimulation. Evidently, people who are sleeping do not awaken easily because of pain (see Foo & Mason, 2003, for a review).

Furthermore, although many studies have yielded significant correlations between pain severity and insomnia (e.g., Atkinson et al., 1988; Haythornthwaite, Hegel, et al., 1991; Wilson et al., 1998), the magnitude of the relationship is typically modest. For instance, Wilson et al. (1998) found that pain accounted for less than 20% of the variance in most diary measures of sleep continuity, and Smith et al. (2000) reported that pain severity was not a significant predictor of global sleep disturbance as measured by the PSQI. Therefore, the hypothesis that pain *per se* is the sole cause of an individual's sleep disturbance is not fully supported by the evidence. Chronic pain can certainly be construed as a risk factor for insomnia, but it is apparently not the only relevant variable.

Insomnia causes pain. In addition to a causal pathway that posits that pain causes insomnia, some investigators have suggested that the opposite is also true, and that insomnia causes pain. In fact, there is evidence to suggest that insomnia may increase the experience of pain, either physiologically or perceptually. This hypothesis was first raised by Moldofsky and colleagues, who were interested in investigating the relationship between disturbed sleep and the widespread muscular aching characteristic of fibromyalgia. Moldofsky et al. found that systematic deprivation of stage 4 sleep in healthy volunteers was associated with the development of musculoskeletal pain (Moldofsky & Scarisbrick, 1976; Moldofsky et al., 1975). Similarly, Chiu et al. (2005)

reported that disturbed sleep, in addition to depressive symptoms, were each independently associated with a decreased pain threshold in an experimental manipulation among individuals with chronic pain. Taken together, these studies suggest that part of the association between poor sleep and pain may lie in the fact that insomnia can intensify the pain experience. In fact, there is evidence to suggest that sleep deprivation can also counteract the analgesic effects of pharmacological interventions (see Kundermann, Krieg, Schreiber, & Latenbacher, 2004, for a recent review). However, it should be noted that studies of sleep deprivation among healthy volunteers may not generalize to individuals with chronic insomnia.

Other research has drawn a similar conclusion. As discussed earlier, Haythornthwaite, Hegel, et al. (1991) studied 46 chronic pain patients and found that total sleep time was correlated negatively with pain intensity, while daytime pain intensity was correlated with difficulties initiating sleep the following night. They thus concluded that pain and insomnia may exert bidirectional influences on one another, with increased pain contributing to insomnia and insomnia increasing pain intensity. Affleck et al. (1996) conducted a 30-day, prospective self-monitoring study and found that poor sleep predicted a more painful day in women with fibromyalgia, thus providing partial support for the notion that insomnia precedes daytime pain rather than follows it. However, Affleck et al. (1996) also found that pain was in turn associated with poorer sleep the following night. They speculated that sleep disturbance leads to fatigue, diminished concentration, and a poor use of coping skills, which in turn can exacerbate pain intensity and reduce the ability to distract oneself from pain.

In summary, pain and insomnia may exert bidirectional influences, and there is evidence for a self-perpetuating cycle in which pain, poor sleep, and affective distress interact with one another. It appears likely that insomnia is not merely a byproduct of pain, but may exacerbate it in a reciprocal fashion.

Affective distress mediates the pain-insomnia link. As discussed previously, depression is another common, albeit not universal, feature of chronic pain, and it is well known that depression is associated with poor sleep. It can be argued, therefore, that insomnia among chronic pain patients is attributable to comorbidity with elevated affective distress. In support of this hypothesis are the findings that symptoms of anxiety and depression are more severe among chronic pain patients with insomnia when compared to those without insomnia (Atkinson et al., 1988; Korszun et al., 2002; Pilowsky et al., 1982; Shaver et al., 1997; Wilson et al., 2002). In fact, Haythornthwaite, Hegel, et al. (1991) found that self-reports of anxiety and depression were more consistently and uniformly correlated with measures of disturbed sleep than was pain intensity.

These findings notwithstanding, affective distress does not appear to function as a straightforward mediator in the pain–insomnia relationship. For instance, there is evidence to suggest that many chronic pain patients without anxiety or depressive disorders (assessed via structured interview) nevertheless experience significant sleep impairment (Currie et al., 2000; Wilson et al., 1998). Furthermore, although Smith et al. (2000) found that depressive symptoms were significantly correlated with pain and sleep complaints in their sample of chronic pain patients, regression analyses revealed that depressive symptoms and pain severity were not significant predictors of the severity of

the sleep disturbance. And as discussed earlier, Wilson et al. (2002) reported that insomnia severity was a significant contributor to the prediction of pain severity even after depression was held constant. Thus, the insomnia observed among patients with chronic pain is apparently not simply an artifact of co-morbid depression, but more research is necessary in this area, particularly with regard to those patients who are diagnosed with clinical depressive disorders. Depression alone, in the absence of chronic pain, is associated with a number of marked changes in sleep, but little sleep research has been conducted that has had a combined focus on both depression and chronic pain.

Cognitive-behavioural model of insomnia secondary to chronic pain. As indicated in Table 2, according to the DSM-IV [American Psychiatric Association (APA), 1994], a diagnosis of primary insomnia is given if insomnia symptoms do not co-occur with another mental disorder (e.g., depression), and are not directly attributable to a physiological substance or to a general medical condition (e.g., chronic pain). Thus, insomnia observed among chronic pain patients with or without depression is, arguably, a secondary phenomenon. It is possible that some individuals with long-standing insomnia later go on to develop chronic pain, but even then it is rare for them to report that pain does not at least exacerbate the problem. For instance, Morin et al. (1998) found that greater than 85% of their sample of poor sleepers reported that their insomnia developed concurrently with or after the development of chronic pain.

When insomnia is assigned a secondary status, it is presumed that it is caused by the primary problem; therefore, the most reasonable treatment for a secondary insomnia would be to treat the primary problem. However, there are some medical conditions, like chronic pain, in which the underlying pathology may be untreatable, and the patient

requires assistance in coping with the condition rather than curing it. Recently some investigators have begun to speculate that the cognitive-behavioural model may have some applicability to these circumstances as well, which opens the door to CBT as a treatment for insomnia that occurs secondarily to painful medical illness. The rationale for this extension of the CBT model is inherent in the consideration of 'Perpetuating Factors', depicted in Figure 2. As mentioned earlier, these perpetuating factors reflect either conditioning or coping phenomena. Conditioning can be viewed as occurring in a relatively passive way; an individual need only lie awake for extended periods to reduce the association of the bed with sleep, and increase its association with alert wakefulness. These processes should occur regardless of the initial cause of the insomnia.

The other component of the CBT model encompasses maladaptive behaviours that are used in order to cope with the effects of sleep loss. Again, however, people are likely to try to cope with these effects, regardless of the initial cause of the sleep loss. It would seem, therefore, that the stage is set for any type of secondary insomnia to acquire a learned, psychophysiological component, as long as the period of sleep loss is extended over time.

Several researchers have adapted the cognitive-behavioural model of primary insomnia to the case of insomnia secondary to chronic pain (Currie, 1998; Currie et al., 2000; Edinger, Wohlgemuth, Krystal, & Rice, 2006; Rybarczyk et al., 2005; Smith & Haythornthwaite, 2004; Smith et al., 2000). In this context, the development of pain serves as the initial, precipitating event that leads to the development of acute insomnia, in a manner that parallels the development of primary insomnia. The individual may fail to adapt to the pain and the sleep disturbance for a variety of reasons, such as continuing

pain and its associated stressors, or the development of maladaptive sleep-related behaviours, such as spending excessive amounts of time in bed (often in an attempt to alleviate pain, and/or to 'catch up' on lost sleep). These actions presumably result in a state of wakefulness conditioned to the sleep situation. In support of this model, Smith et al. (2000) found that presleep cognitive arousal was the primary predictor of self-reported sleep quality, whereas pain severity was not.

This model also presumes that the factors that perpetuate the insomnia are similar in both primary and secondary sleep disorders. As with primary insomnia, therefore, these maintaining factors could become the focus of psychological intervention for people with secondary insomnia, using a treatment that targets maladaptive sleep-related cognitions and behaviours.

Few studies have examined cognitive-behavioural treatments of insomnia secondary to chronic pain, and only one single-case study (Morin, Kowatch, & O'Shanick, 1990) examined it in the context of co-morbid major depression. Two small-scale studies evaluated the effectiveness of stimulus control (Morin, Kowatch, & Wade, 1989) and sleep restriction (Morin et al., 1990; Morin et al., 1989) on insomnia secondary to chronic pain (Morin et al., 1989) and chronic pain with major depression (Morin et al., 1990). Although these studies showed meaningful improvements associated with these therapies, they unfortunately involved very small sample sizes ($n_s = 1$ and 3). Recent work by Currie and colleagues (Currie, Wilson, & Curran, 2002; Currie et al., 2000) has provided more compelling evidence for the effectiveness of CBT for insomnia among patients with chronic pain. They found that patients with insomnia secondary to chronic pain who were treated with multicomponent CBT showed significantly greater improvement on

questionnaire, diary, and ambulatory monitoring measures of sleep disturbance, compared to a waiting-list control group. In addition, treatment gains were well maintained at three months' follow-up. More recently, Edinger et al. (2006) conducted a randomized clinical trial of CBT for insomnia in 42 patients with fibromyalgia. They found that participants treated with CBT for insomnia experienced an average reduction of 48% in time awake after sleep onset, which was significantly better than comparison participants who had received sleep hygiene therapy as a stand-alone treatment. Fifty-seven percent of participants treated with CBT evidenced significant sleep improvements after treatment, which was significantly higher than those who had undergone sleep hygiene therapy only. In addition, significant pain reductions were observed in the CBT group and a subset of participants in the sleep hygiene group who had elected to incorporate aspects of CBT in their treatment. However, Currie et al. (2000, 2002) and Edinger et al. (2006) did not include patients with co-morbid major depression. This further obviates the need for more research to evaluate cognitive and behavioural determinants of insomnia secondary to chronic pain, among patients with and without co-morbid major depression.

Depression and Insomnia

Depression among insomniacs. As mentioned earlier, the frequent comorbidity of depression and insomnia is also well-established within the literature. In fact, insomnia is a cardinal feature of depression, and it is used in the diagnosis of depressive disorders (APA, 1994). Table 4 presents prevalence estimates of major depression, diagnosed according to DSM or RDC criteria, among individuals with insomnia. The average prevalence estimate of major depression among insomniacs recruited from the

Table 4. Prevalence Estimates of Major Depressive Disorder Among Individuals with Insomnia

Study	n	Prevalence Estimate (%)
<u>Community Participants:</u>		
Breslau et al. (1996)	1,007	31.1
Ford & Kamerow (1989)	7,954	14.0
Mellinger et al. (1985)	3,161	21.0
Ohayon et al. (1998)	5,622	18.2
Roberts et al. (2000)	2,370	19.8
Vollrath et al. (1989)	456	25.4
Weissman et al. (1997)	7,113	25.0
Average:		22.1
<u>Primary Care Patients:</u>		
Hohagen et al. (1993)	2,512	28.7
Simon & Von Korff (1997)	373	30.5
Average:		29.6 %

community is 22.1%. Not surprisingly, when depression-spectrum disorders such as dysthymia, recurrent, brief depression (Vollrath, Wicki, & Angst, 1989) and subthreshold depression (Simon & Von Korff, 1997) are also considered, prevalence estimates increase by an additional 7% (Vollrath et al., 1989) to 29% (Simon & Von Korff, 1997). In terms of primary care patients with insomnia, the prevalence estimates for major depression are somewhat higher (29.6%, on average) than those for individuals recruited from the community.

In general, studies have shown that insomnia and depression are consistently correlated (e.g., Bixler et al., 2002; Breslau et al., 1996; Hohagen et al., 1993, 1994; Moul et al., 2002; Ohayon & Roth, 2003), that individuals with insomnia are at a substantially elevated risk for major depression (e.g., Breslau et al., 1996; Mellinger et al., 1985; Ohayon & Roth, 2003; see Riemann & Voderholzer, 2003, for a review), and that depressed individuals report persistent insomnia significantly more often than nondepressed (e.g., Mallon, Broman, & Hetta, 2000; Ohayon & Roth, 2003). For instance, Mellinger et al. (1985) found that the risk of major depression was more than three times greater among those with serious insomnia compared to those without serious insomnia. Ford and Kamerow (1989) found that, of all the psychiatric disorders, insomnia of two or more weeks' duration was most strongly related to major depression. Breslau et al. (1996) found that the strongest association between insomnia and psychiatric disorders was observed for major depression, even when the diagnosis was based on symptoms other than sleep disturbance. Furthermore, they found that odds ratios for major depression associated with insomnia were markedly higher than for those of other psychiatric diagnoses.

There is also evidence to suggest that insomniacs without major depressive disorder nevertheless experience significant depressive symptomatology. For instance, Weissman, Greenwald, Nino-Murcia, & Dement (1997) found that patients with primary insomnia, regardless of the fact that they had not been diagnosed with a psychiatric disorder, were almost five times as likely to have had depressive symptoms in the past year when compared to individuals with no insomnia and no psychiatric disorder.

Insomnia among depressed individuals. Surprisingly, only a few studies have provided prevalence estimates of insomnia among patients who have been diagnosed with depressive disorders. Ohayon, Shapiro, & Kennedy (2000), in a telephone survey of Toronto adults, reported that 75.5% of those individuals diagnosed with a mood disorder also complained of insomnia. Mallon et al. (2000) found that 85.6% of their sample of self-described depressed individuals reported insomnia (with 52.6% reporting persistent insomnia). Perlis, Giles, Buysse, Thase, et al. (1997) reported that 65% of their sample of patients with major depression also had one or more severe sleep complaints (38% had initial insomnia, 39% had middle insomnia, and 41% experienced early morning awakening). Ohayon (1997) reported that 55.6% of their sample of French adults with major depression also complained of insomnia. Overall, then, it appears that about 14% to 32% of community residents who complain of insomnia also meet diagnostic criteria for major depression. Alternatively, 65% to 86% of people who complain of depression suffer from insomnia.

Insomnia as a precursor to depression. It is conceivable that the common co-occurrence of depression and insomnia may simply reflect the fact that insomnia can be a symptom of depression. However, there is compelling evidence to suggest that insomnia

may function as a precursor to, or prodrome of, the depressive disorder. Longitudinal studies have shown significantly increased rates of new cases of depression among individuals reporting insomnia at baseline and follow-up (Breslau et al., 1996; Ford & Kamerow, 1989; Roberts et al., 2000), and at baseline only (Chang, Ford, Mead, Cooper-Patrick, and Klag, 1997; Livingston et al., 1993; Mallon et al., 2000; Weissman et al., 1997), with a median follow-up period of up to 34 years (Chang et al., 1997; see Riemann & Voderholzer, 2003, for a recent review). For instance, Breslau et al. (1996) found that the gender-adjusted relative risk for a new episode of major depression was four times greater for individuals with a lifetime history of two or more weeks of insomnia than for those without insomnia. Livingston et al. (1993) conducted path analyses and found that causal pathways were observed from insomnia to depression, whereas the reverse (from depression to insomnia) was not the case.

In some studies, the depression-insomnia association has been confined to female participants (Dryman & Eaton, 1991). For example, Mallon et al. (2000) found that self-reported insomnia symptoms were significant predictors of depression 12 years later, but this effect only held for women. Insomnia has also been implicated in the recurrence of depression. In another longitudinal study, two or more weeks' of sleep disturbance was found to be significantly associated with the recurrence of major depression among formerly depressed patients in remission (Perlis, Giles, Buysse, Tu, & Kupfer, 1997).

Nevertheless, although insomnia has been shown to frequently precede the onset of major depression, the depression-insomnia relationship is not clear-cut. For instance, although Roberts et al. (2000) found that insomnia was a significant predictor of major depression, other symptoms (psychomotor agitation or retardation, anhedonia, feelings of

worthlessness, depressed mood and thoughts of death) were better predictors of future depression than sleep disturbance. Additionally, although Chang et al. (1997) found that the relative risk for developing clinical depression was significantly elevated (approximately double) among their sample of men reporting insomnia at baseline, the majority of participants who subsequently developed depression had not experienced insomnia at baseline.

The elucidation of the specific nature of the relationship between depression and insomnia carries with it implications for both theory and treatment. As indicated earlier, Ford and Kamerow (1989) and Roberts et al. (2000) found that insomnia at a single assessment alone was not significantly related to subsequent depression, thus suggesting that only long-term, persistent insomnia is associated with increased risk. This also suggests that the successful resolution of insomnia (e.g., through early intervention) may prevent the development of depression, and argues for the utility of monitoring the sleep of individuals at risk for new or recurrent depressive episodes (Breslau et al., 1996; Dryman & Eaton, 1991; Livingston et al., 1993). These studies also provide evidence against the notion that insomnia is an epiphenomenon of depression (Ford & Kamerow, 1989), and implies that disturbed sleep may play a critical role in the pathogenesis of depression for some individuals (Perlis, Giles, Buysse, Thase et al., 1997).

The correlates of depression and insomnia. As indicated earlier, there is some research to suggest that the comorbidity of depression and insomnia is particularly pronounced among women (Dryman & Eaton; Mallon et al., 2000). This is perhaps not surprising, given the fact that female gender is frequently a significant correlate of insomnia and that women are more frequently diagnosed with depression than men

(Nolen-Hoeksema, 1987). In a large-scale telephone survey of 5,622 French adults who were interviewed about specific DSM-IV disorders, Ohayon et al. (1998) found that significantly more women than men were diagnosed with insomnia related to a depressive disorder and depressive disorder with insomnia symptoms than with primary insomnia or isolated insomnia symptoms. Elderly respondents were also significantly overrepresented in the isolated insomnia group. Ohayon et al. (1998) also found that respondents with insomnia secondary to a mental disorder (depressive or anxiety disorder, in this case) generally had a longer history of insomnia complaints and were younger than those with depressive or anxiety disorders accompanied by subthreshold insomnia symptoms. Additionally, subjects with insomnia related to a depressive disorder experienced more negative repercussions (e.g., sleep drunkenness, feeling restless at awakening, difficulties getting started) than did any other category of sleep disorder.

Suicidal behaviour is another frequent and important correlate of depression and insomnia, thus making insomnia an issue worthy of specific attention and intervention. For instance, Agargun, Kara, and Solmaz (1997) found that patients with disturbed sleep scored significantly higher than individuals without sleep disturbance on the suicide subscale of the Schedule for Affective Disorders. Global and partial insomnia have been shown to be significant risk factors for suicide within one year in several large-scale longitudinal studies (Clayton, 1993; Fawcett, Clark, & Busch, 1993; Fawcett et al., 1990, Hall, Platt, & Hall, 1999; see Singareddy & Balon, 2001, for a review).

PSG studies of depression and insomnia. Benca, Obermeyer, Thisted, and Gillin (1992) conducted a meta-analysis of 177 studies (incorporating 7,151 insomniac and control subjects) on sleep and psychiatric disorders and found that the PSG characteristics

of patients with affective disorders, relative to other types of mental health problems, differed most frequently and significantly from those of controls. Approximately 90% of patients with major depression show polysomnographic evidence of sleep disturbance (Kupfer, 1999; Reynolds, 1987). In general, the sleep of depressed individuals is characterized by several features: (1) changes in sleep continuity (increased sleep latency, increased number of awakenings, including early morning awakenings); (2) alterations in non-REM sleep (decreased slow-wave sleep, decreased delta sleep in the first non-REM period; (3) alterations of REM sleep (decreased REM latency [less than 65 minutes]), increased REM sleep in first half of the night, and increased REM density) (Reynolds & Kupfer, 1987; Winokur & Reynolds, 1994, cited in Kupfer, 1999). In contrast, the sleep of primary psychophysiological insomniacs generally show no alterations in sleep architecture or REM abnormalities other than delayed sleep onset and frequent awakenings (Diagnostic Classification Steering Committee, 1990; Kales & Kales, 1987).

Perlis, Giles, Buysse, Thase, et al. (1997) found that a collection of neurovegetative, cognitive, and affective symptoms was related to EEG sleep disturbance among patients with major depression. Furthermore, this relationship was unidimensional in nature, thus suggesting that EEG sleep disturbance is most related to the broad symptoms of depression (depressed mood, loss of appetite, disturbed sleep, feelings of guilt) rather than to any specific subset of depressive symptoms (neurovegetative, cognitive, affective).

In addition, there is evidence to suggest that these PSG characteristics are more stable and trait-based than episodic in nature (Rush et al., 1986), that they tend to be maintained throughout the course of depression, and that some persist even after remission and

medication withdrawal (with the exception of sleep continuity, sleep-onset REM periods, and phasic REM sleep, which generally tend to normalize) (see Buysse & Nofzinger, 1994, for a detailed description).

Furthermore, several PSG features have been implicated in the recurrence of depression. Giles et al. (1987, cited in Buysse & Nofzinger, 1994) reported that reduced REM sleep latency was associated with recurrence of depression up to 24 months after successful treatment with psychotherapy and pharmacotherapy. Similarly, Kupfer et al. (1990, cited in Buysse & Nofzinger, 1994) found that remitted patients who nevertheless showed a reduced delta ratio had a significantly reduced latency to depression recurrence during a 3-year follow-up than individuals with normal delta activity.

Chronic Pain, Depression, and Insomnia

Several studies have highlighted the frequent co-occurrence of depression and insomnia among chronic pain patients. With few exceptions, these studies have relied on self-reports of depressive symptomatology and disturbed sleep rather than structured, criterion-based diagnostic assessments. In general, most of these studies have confirmed that depressive symptoms and poor sleep are correlated in patients with chronic pain (Atkinson et al., 1988; Becker et al., 1997; Morin et al., 1989, 1990, 1998; McCracken & Iverson, 2002; Nicassio et al., 2002; Nicassio & Wallston, 1992; Palermo & Kiska, 2005; Pilowsky et al., 1985; Riley et al., 2001; Robbins, 1995; Shaver et al., 1997; Smith et al., 2000; Wittig et al., 1982).

In fact, there is evidence to suggest that the combination of chronic pain, depressive symptoms, and insomnia is related to increased risk of suicide. Other estimates of suicidal ideation among such groups have ranged from 6.5% (Fisher,

Haythornthwaite, Heinberg, Clark, & Reed, 2001) to 24% (Smith, Perlis, & Haythornthwaite, 2004). Researchers have also noted that suicidal ideators had significantly higher levels of pain (Fisher et al., 2001; Smith et al., 2004) and insomnia (Smith et al., 2004; see Tang & Crane, 2006, for a review). Regression analyses have also shown suicidal ideation to be associated with pain, independent of depression severity (Smith et al., 2004).

There is a paucity of research, however, devoted to the comorbidity of insomnia with diagnosed major depressive disorder (as opposed to symptom self-reports on measures of general distress) within a chronic pain population. Harman et al. (2002) conducted PSG assessments over four nights on ten participants with chronic low back pain (4 of whom had comorbid major depression), and 11 comparison participants. Individuals with chronic low back pain reported significantly higher levels of sleep disturbance and pain relative to controls. The groups did not differ regarding sleep duration and sleep architecture; however, individuals with chronic pain evidenced lower sigma power in their PSG recordings, suggesting that suboptimal sensorimotor gating in the central nervous system may contribute to disturbed sleep. Further, participants with both chronic pain and comorbid major depression showed increased occipital delta, occipital and central alpha, and beta activity in their sleep recordings than the nondepressed group with chronic low back pain. Thus, the PSG profiles of individuals with chronic pain and comorbid major depression may differ in certain ways from those without comorbid depression. However, more research, with larger sample sizes, is necessary in order to elucidate these complex relationships.

Korszun et al. (2002) compared 16 FM patients to 6 FM patients with comorbid major depression, 9 non-pain participants with recurrent major depression, and 28 healthy controls. They found that participants with FM alone and major depression alone showed comparable levels of sleep disturbance, both of which were significantly more impaired than normal controls. However, participants with FM and comorbid major depression had the most problematic sleep, including significantly more daytime naps and significantly more activity during the night.

Wilson et al. (2002) studied a substantially larger sample of 150 consecutive patients at a multidisciplinary pain clinic and found that rates of insomnia were significantly higher among patients with major depression when compared to those without. In addition, clinicians' ratings of insomnia severity showed that the sleep disturbance among depressed insomniacs was more extreme than the sleep disturbance of insomniacs without major depression. Wilson et al. (2002) found that 25% of patients suffered from comorbid major depression and insomnia, while 39% complained of insomnia but were not diagnosed with major depression, 5% had major depression without insomnia, and 31% were diagnosed with neither depression nor insomnia. Therefore, 30% of all respondents met diagnostic criteria for major depression; of these, 84.4% also had clinically significant insomnia. Further, of the 70% of the sample not diagnosed with major depression, 55.2% nevertheless suffered from clinically significant insomnia. Thus, insomnia can be said to affect a substantial portion of chronic pain patients, but it appears to be more prevalent and more severe among those diagnosed with major depression.

Wilson et al. (2002) also found that chronic pain patients with insomnia and major depression scored significantly worse than noninsomniacs on several measures of affective distress and pain severity. Although patients with insomnia only reported lower levels of disability than those with both major depression and insomnia, they nonetheless reported worse functioning on some measures of pain severity and distress than noninsomniac patients.

Although Wilson et al.'s (2002) study is the most comprehensive to date on depression and insomnia within a chronic pain population, several key issues warrant further consideration in future research. Wilson et al. investigated insomnia as a symptom rather than a diagnosable disorder in its own right; they also relied on retrospective self-ratings of 'typical' sleep rather than daily diary assessments or objective measures of sleep parameters.

Summary

Chronic pain, major depression, and insomnia are all common problems individually, but they also frequently co-occur. Each can be conceptualized within a cognitive-behavioural framework, but they have seldom been investigated together in a formal manner. CBT for insomnia appears to be applicable to pain patients; however, its applicability to depressed patients with insomnia is unclear (Chesson et al., 1999; Smith et al., 2005; Smith & Perlis, 2006), and to depressed chronic pain patients even more so. To date, the evidence suggests that it would be premature to treat depressed chronic pain patients with CBT for insomnia, until the depression itself has been addressed. However, it might be possible to incorporate CBT for insomnia directly into a multidisciplinary treatment for chronic pain more generally. It would be worthwhile in this context to have

a better understanding of the similarities and differences in the sleep behaviour of chronic pain patients with and without major depressive disorder. As noted, the only available studies have been conducted with very small samples and crude assessments of insomnia based upon retrospective reports of how people 'typically' sleep. Moreover, the dimensions of sleep that have been assessed have not been selected on the basis of a conceptual grounding of the CBT model of insomnia, which would be particularly relevant for informing eventual treatment research.

More evidence for the relevance of this treatment is necessary before attempting clinical trials with this population, particularly in light of the fact that CBT for insomnia, especially in the initial stages, can be very disruptive of individuals' sleep habits (e.g., patients are usually instructed to make changes in their sleep routines, including staying up later than usual and avoiding naps). Unfortunately, since many individuals undergoing multidisciplinary treatment for chronic pain meet the criteria for major depressive disorder, solid empirical support for the applicability of this treatment is necessary before generalizing it to individuals with fragile mental health (Smith et al., 2005).

The present study therefore involved a detailed examination of the sleep habits, cognitions, and behaviours of insomniac chronic pain patients with and without major depressive disorder. Criterion-based diagnostic assessments were used to categorize depression and insomnia, and sleep was investigated using a multi-method approach that incorporated questionnaires and daily diary measures of pain, sleep, and mood.

Rationale for the Present Study

The cognitive and behavioural determinants of insomnia among chronic pain patients with and without depression warrant empirical investigation for a number of reasons.

Although Morin et al. (1999) argued for the applicability of behavioural treatments for insomnia in patients with comorbid psychiatric disorders, in research and in practice, it is common to exclude depressed patients from behavioural interventions for insomnia (e.g., Currie et al., 2000; Smith et al., 2005; Smith & Perlis, 2006; Stepanski & Rybarczyk, 2006). Moreover, in the case of chronic pain, there has never been a study with an adequate sample size that used structured clinical diagnostic interviews for major depression and insomnia, in addition to daily diary methods (rather than historical reports of 'typical' behaviours), in the context of the specific components of the CBT model of insomnia. If insomniac patients with comorbid major depression simply represent the most extreme group in terms of negative sleep-related behaviours, then their exclusion from behavioural treatments for insomnia would perhaps be unwarranted, unless clinical considerations other than disturbed sleep mitigate against their inclusion (e.g., suicide attempts at night during sleep restriction (Smith et al., 2005; Smith & Perlis, 2006). Furthermore, intervening in problematic cognitions and behaviours could potentially help both depression and insomnia simultaneously.

Hypotheses of the Current Study

Hypothesis 1. It was hypothesized that chronic pain patients with major depression would report higher levels of pain and functional disability when compared to patients without major depression. This would be reflected in retrospective (Modified Pain History Questionnaire, including the Disability subscale) and daily diary (Pain Severity Ratings Scales) self-report measures. This hypothesis sought to replicate earlier research highlighting the significant, positive relationships reported in the literature regarding estimates of major depression and increased pain severity (Benjamin et al.,

1988; Currie & Wang, 2004; Elliott et al., 2003; Haley et al., 1985; Haythornthwaite, Sieber, et al., 1991), and self-reported disability (Currie & Wang, 2004; Elliott et al., 2003). In this sense, the study will investigate whether the effects of pain and major depression have effects on sleep that are additive to one another. However, the current study extends the findings of the previous studies in that it included daily diary assessments of pain severity rather than solely retrospective reports.

Hypothesis 2. It was hypothesized that patients with major depression would report worse sleep, and exhibit more problematic sleep-related behaviours than patients without major depression. Specifically, it was hypothesized that patients with major depression would be more likely to receive a diagnosis of insomnia, and would report significantly higher levels of insomnia severity within the interview portion of the study. It was also expected that depressed patients would exhibit more impaired sleep according to both retrospective (i.e., the PSQI) and prospective (i.e, the DSD, time spent napping) self-reports of sleep parameters. This is based upon previous literature documenting significantly poorer sleep among chronic pain patients with comorbid major depression (Korszun et al., 2002; Wilson et al., 2002), and is consistent with the fact that poor sleep can be a diagnostic feature of major depression (e.g., APA, 1994). However, the present study sought to elaborate upon previous research by examining pain, sleep, and mood from an *in situ*, daily diary perspective.

In addition to experiencing poor sleep, it was expected that patients with major depression would differ significantly from their nondepressed counterparts on behaviours associated with chronic insomnia. This is consistent with the work of Morin and colleagues, and the theoretical role of such behaviours in perpetuating insomnia

(Morin, 1993; Spielman et al., 1987), and the findings of Woodley and Smith (2006), who reported significant, positive relationships between sleep-incompatible behaviours and depressive symptoms. However, the current study represents an extension of these theories to participants with major depression. This would be reflected in retrospective reports (i.e., Sleep Hygiene Usual Practice Scale) and daily diary (i.e., Sleep Hygiene Daily Diary) self-report measures. The current study also expanded upon previous research in that it examined these behavioural reports of sleep-related behaviours specifically within a day-to-day context, and with novel adaptations of assessment measures traditionally used in a retrospective manner.

Hypothesis 3. In keeping with the cognitive-behavioural conceptualization of insomnia (Morin, 1993; Spielman et al., 1987), it was further hypothesized that depressed patients would differ significantly from nondepressed participants in terms of maladaptive cognitions (e.g., attitudes, knowledge, and beliefs) about sleep. This is consistent with the work of Woodley and Smith (2006), who found that dysfunctional attitudes and beliefs about sleep were associated with depression symptoms. This hypothesis would be assessed retrospectively (via the DBAS, Sleep Hygiene and Caffeine Knowledge) and prospectively (PSAS, including cognitive, somatic and total arousal).

Exploratory analyses. A series of exploratory investigations was also proposed, which involved various *t*-tests and correlational analyses. The differences between participants with and without major depression were compared on mood-related variables; this served to confirm the validity of the diagnostic assessments. The correlates of global insomnia severity among both groups were analyzed and compared, because the existent literature tended to exclude participants with chronic pain and comorbid major

depression (e.g., Currie et al., 2000; Edinger et al., 2006; Korszun et al., 2002). Finally, in order to address specifically the potential applicability of CBT for insomnia within this population, correlation coefficients were computed between major depression, depressive symptoms and the most salient cognitive-behavioural targets of CBT for insomnia. This was undertaken with the intention of identifying specific therapeutic aspects of CBT for insomnia in future research involving clinical trials among patients with chronic pain, insomnia, and comorbid major depression.

Method

Participants

Seventy-four patients were recruited through the Pain Clinic of the Rehabilitation Centre. This is a multidisciplinary assessment clinic that specifically addresses the problem of chronic musculoskeletal pain. In general, patients referred to this service have typically exhausted all other medical options for obtaining sustained pain relief, but are continuing to have difficulty coping on a long-term basis with a persistent pain complaint. The Pain Clinic provides a specialized assessment service, often in a multidisciplinary format involving physiatry, physiotherapy, and psychology, with the goal of identifying an integrated treatment plan that will aid the individual in achieving and maintaining an optimal level of functioning.

Patients who have been referred to the Pain Clinic at The Rehabilitation Centre complete a questionnaire battery before their initial appointment. Based on these responses, triage judgements are made by the clinic staff as to whether a solely medical or a multidisciplinary assessment is necessary. This decision is made based on patient reports of psychological distress, social concerns, functional limitations, and/or excessive medication usage. Many of these patients are considered appropriate for participating in a structured pain management program. If so, the initial point of entry into the treatment is through an orientation session.

Procedure

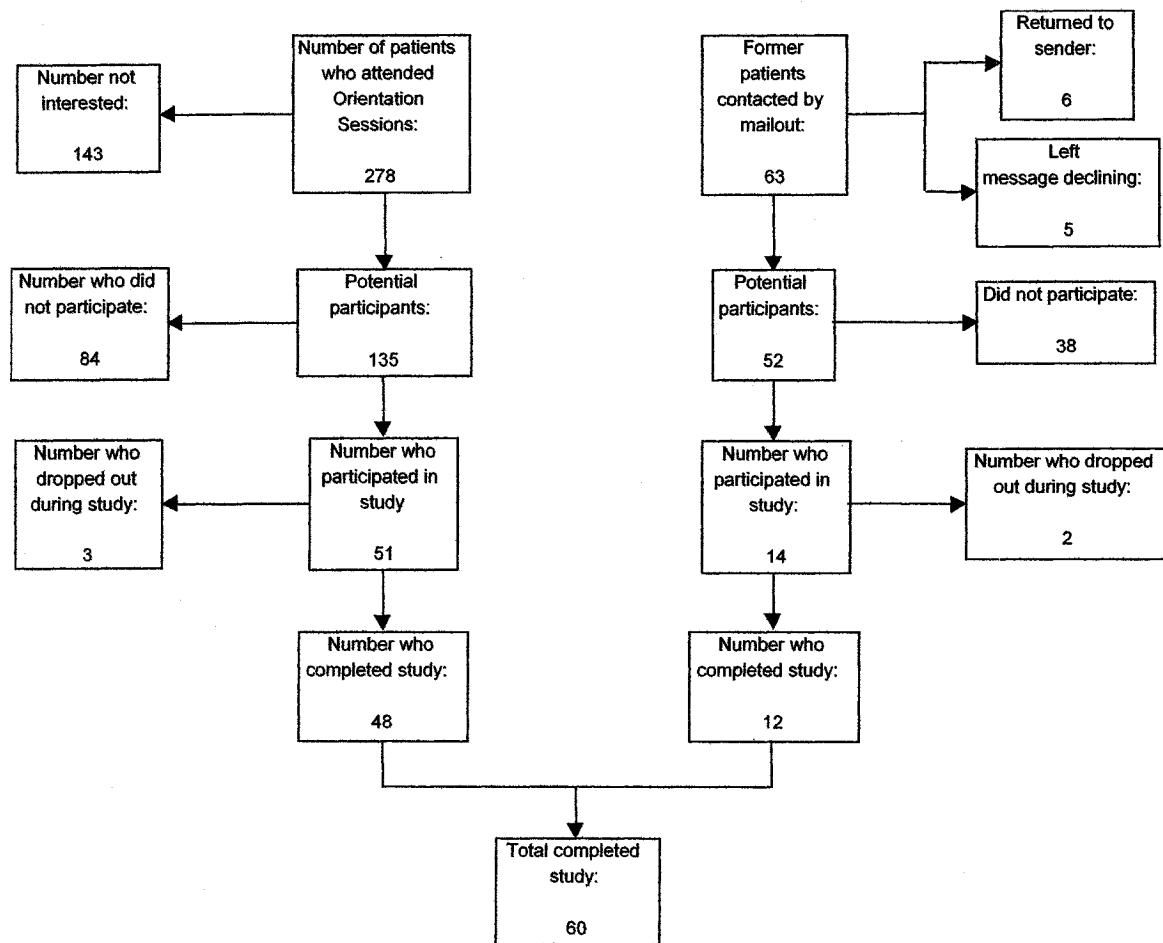
The orientation sessions were held every two weeks, and were typically composed of eight to nine individuals. Ms. Emery or Dr. Wilson approached a total of 278 prospective participants at the beginning of these sessions, providing an information sheet about the

study (see Appendix A) in addition to an individual sign-up sheet for each person who was interested in learning more (see Appendix B). Fifty-one individuals recruited from orientation sessions were successfully contacted and agreed to participate in the study; however, three dropped out after the interview and failed to complete the entire protocol (see Figure 3).

In addition to the recruitment of patients before treatment, patients seen at The Rehabilitation Centre within the past year were also approached for participation. These individuals ($n = 63$) were mailed a letter explaining the purpose of the study and a brief overview of what their participation would entail (see Appendix C). Six of these letters were returned because the address on file was not current. Five patients notified The Rehabilitation Centre by telephone that they did not wish to participate. Fourteen individuals from the mailout agreed to participate; however, 2 individuals from this recruitment pool did not finish the study. We did not have Research Ethics Board approval to pursue individuals who refused to participate. Therefore, we cannot comment on how they may have differed from those who volunteered.

Ms. Emery contacted prospective participants by telephone and further explained the purpose and procedures of the study. At this point, patients were also screened to ensure that they did not have any comorbid medical conditions that could affect sleep (e.g., pregnancy, cancer, obstructive lung disease, acquired brain injury, or other neurological conditions); patients reporting concurrent conditions of this nature were excluded from the study ($n = 5$). Only patients under the age of 60 were invited to participate, in order to limit the influence of age-related factors on sleep continuity (Morin, 1993); five interested participants were excluded due to age.

Figure 3. Flowchart of Participant Recruitment



The script for the telephone recruitment is provided in Appendix D. Once verbal consent was obtained, a personal interview was arranged, either at the patient's home or at The Rehabilitation Centre. Participants choosing the latter option had their parking or other transportation expenses reimbursed. A maximum of three attempts was made to reach prospective participants before the individual was excluded from the study.

Immediately before the interview, participants were provided with an additional information sheet about the study (see Appendix A) and signed 2 acknowledgements of informed consent, one of which the participant retained (see Appendix E). After the interview was conducted, participants were provided with a set of questionnaires to be completed at their convenience. They were also provided with four sets of daily diary measures, which they were asked to complete each morning and throughout the day and at bedtime. Each participant was instructed to complete the questionnaires and daily diary measures without assistance or interference from others.

Measures

A detailed schematic of the assessment measures employed in the current study is presented in Table 5.

Diagnostic Interviews

Participants underwent an in-depth personal interview to diagnose specific psychological syndromes, including sleep disorders. The interviews were audiotaped, in order to permit the assessment of interrater reliability.

Primary Care Evaluation of Mental Disorders (PRIME-MD). This semi-structured diagnostic interview protocol (Spitzer et al., 1994) is based on DSM-IV criteria (APA, 1994). The PRIME-MD interview consists of two components. First, the participant was

Table 5. Assessment Measures by Method of Administration and Research Hypothesis**Interview Assessment****Mood:**

Primary Care Evaluation of Mental Disorders

Sleep and Sleep-Related Behaviours:

Structured Interview for Sleep Disorders

Questionnaire/Retrospective Assessment**Demographics, Pain and Disability:**

Modified Pain History Questionnaire – Demographics and retrospective pain severity

Disability Subscale

Mood:

BDI-II

Sleep and Sleep-Related Behaviours:

Pittsburgh Sleep Quality Index

Sleep Hygiene Usual Practice Scale

Sleep-Related Cognitions:

Dysfunctional Beliefs and Attitudes About Sleep Scale

Sleep Hygiene Knowledge Scale

Caffeine Knowledge

Daily Diary Assessment**Pain and Disability:**

Pain Severity Ratings Scales

Mood:

Positive and Negative Affect Schedule

Table 5 (cont'd). Assessment Measures by Method of Administration and Research Hypothesis

Daily Diary Assessment (cont'd):

Sleep and Sleep-Related Behaviours:

Daily Sleep Diary

Napping

Sleep Hygiene Daily Diary

Sleep-Related Cognitions:

Presleep Arousal Scale

asked to complete the Patient Questionnaire (see Appendix F), which is a series of 'yes/no' questions about psychological difficulties experienced within the past month. These questions correspond to the central criterion symptoms of specific mental disorders. Second, the interviewer followed up using a structured interview guide that provided complete coverage of the diagnostic criteria for the disorders in question. The PRIME-MD allowed for the diagnosis of depression-spectrum disorders (e.g., major depression, dysthymia), anxiety disorders [e.g., panic disorder, generalized anxiety disorder (GAD)], and alcohol abuse. Spitzer et al. (1994) reported good concordance between PRIME-MD diagnoses and those of independent mental health professionals using other structured interview protocols ($\kappa = 0.71$ for any diagnosis).

The PRIME-MD originally comprised five modules (mood, anxiety, somatoform disorders, alcohol abuse, and eating disorders). For the present purpose, only the sections corresponding to depression, anxiety disorders, and alcohol abuse were considered relevant. The structured interview was also enhanced using the protocol reported by Wilson et al. (2004). With that method, detailed inquiries were made into core criterion symptoms of anxiety, depressed mood, and loss of interest or pleasure in activities, in order to ensure specific adherence to DSM-IV severity thresholds. This modification has been used in several other studies (Currie et al., 2000; Wilson, Scott, et al., 2000). Currie et al. (2000) reported a rate of interrater agreement of 88% ($\kappa = 0.71$).

Structured Interview for Sleep Disorders (SIS-D). The SIS-D is a structured interview assessing specific sleep complaints according to the DSM classification system (see Appendix G). Originally developed according to DSM-III-R criteria, Schramm et al. (1993) reported high levels of interrater agreement (average $\kappa = 0.85$) and good

concordance with clinical judgements based on PSG recordings. Using a version of the SIS-D adapted to DSM-IV criteria, Currie et al. (2000) reported an interrater agreement of 100% for the diagnosis of insomnia. The present study employed an expanded version of the SIS-D adapted for DSM-IV, and screened for a range of dyssomnias: primary insomnia, primary hypersomnia, narcolepsy, breathing-related sleep disorder, circadian rhythm disorders, and dyssomnia NOS. Parasomnias were also evaluated (nightmare disorder, sleep terror disorder, sleepwalking disorder, and parasomnia NOS), in addition to restless legs syndrome and periodic limb movement disorder. It also contained a seven-point Likert scale that provides a global rating of insomnia severity, which ranged from 1 (not at all) to 6 (extreme difficulty sleeping).

Demographics and Medication Usage

Demographic information. Data were collected on relevant demographic characteristics, such as age, sex, ethnic background, preferred language, employment status, and educational background. In addition, patients were asked whether they were currently involved in any legal proceedings related to their chronic pain condition (see Appendix H).

Medication Quantification Scale (MQS). Patients with chronic pain frequently consume prescription and over-the-counter medications, which can have a significant impact on sleep. The MQS (Harden et al., 2005; Steedman et al., 1992; see Appendix I) was employed to provide a method of quantifying medication use over the course of the study, and to evaluate whether or not medication usage differed significantly between the groups. The MQS was designed for use in chronic pain populations. It required the respondent to report his or her daily use of prescription and over-the-counter drugs (e.g.,

antidepressants, sedative-hypnotics, muscle relaxants). For each medication, the respondent listed the medication type, the dosage, and the number of pills taken each day. Scores were calculated based upon weights assigned to the medication class and multiplied by a factor reflecting dosage level (e.g., subtherapeutic or occasional use, the lower half of the therapeutic dose range, the upper half of the therapeutic dose range, or above the accepted therapeutic dose range). The weights were derived from a survey of 187 physicians belonging to the American Pain Society (Harden et al., 2005), and reflect the potential for acute or chronic adverse effects for a given class of drugs. The scores were then summed, thus providing a quantitative score suitable for statistical analysis. The MQS has been shown to be a reliable and valid measure of medication consumption. The Spearman rank-correlation coefficient for MQS scores was 0.98 when 30 medication profiles of chronic pain patients were evaluated by two expert raters. In terms of its validity, 22 health care professionals rated the medication profiles of 88 chronic pain patients; the Spearman rank-correlation coefficient for the mean clinician rating and the MQS rank was 0.76. Further, the MQS has been shown to be sensitive to changes in medication usage associated with treatment gains (Steedman et al., 1992).

Pain and Disability: Retrospective Questionnaire Assessment

Modified Pain History Questionnaire. The Pain History Questionnaire (D'Eon, Petersen, Wilson, & Baldwin, 2004; see Appendix J) is completed routinely by patients at the Pain Clinic as part of their evaluation process; a shortened version was used in the present study. It inquired about the nature of the patient's pain (location, severity, duration, and how it began), and pain severity within the past two weeks (current, average, worst, and least amount of pain), all of which were rated on a 10-point scale,

with 0 denoting no pain, and 10 the worst pain. Previous research has established good test-retest reliability, with r s ranging from 0.65 to 0.90 for retrospective ratings of pain over three weeks. These numerical pain ratings have also been shown to be correlated significantly with the McGill Pain Questionnaire and the Multidimensional Pain Inventory (Jensen, Turner, Romano, & Fisher, 1999).

Disability Subscale of the Pain History Questionnaire. As discussed earlier, the findings of Menefee et al. (2000) highlight the importance of considering physical functioning in studies of chronic pain and insomnia. This questionnaire (IASP Task Force on Records and Data Retrieval, 1995; see Appendix K) asks respondents to rate the degree of difficulty (0 = no difficulty, 1 = slight difficulty, 2 = moderate difficulty, 4 = extreme difficulty, 5 = unable to do) they experience in completing 16 specific everyday tasks (e.g., dress yourself, climb up 5 steps, run errands, shop). The responses are summed, thus yielding a possible range of 0 – 80. The internal consistency of the 16 items in the present study was excellent ($\alpha = 0.88$). In other validation research, scores on this scale have been found to correlate significantly with the General Activity dimension of the Multidimensional Pain Inventory (D'Eon et al., 2004, May).

Pain and Disability: Prospective Daily Diary Assessment

Pain Severity Ratings. On the daily diaries, pain severity was measured with 4 items scored on 0 to 10 numeric rating scales, as reported by Jensen and McFarland (1993; see Appendix L). Specifically, the respondent was asked to rate pain severity on a scale of 0 ('no pain') to 10 ('pain as bad as could be') at four times throughout the day (first thing in the morning, lunchtime, suppertime, and bedtime). Respondents were asked to complete this measure over four days, thus yielding 16 data points over a four-day

period. Jensen and McFarland (1993) have shown that multiple ratings (i.e., at least 12) of pain severity over successive days result in estimates of average pain ratings that have excellent psychometric properties (i.e., internal consistency, test-retest stability, and validity coefficients of 0.90 or more) among chronic pain patients. The current study yielded a Cronbach's alpha value of .97, indicating excellent internal consistency.

Sleep and Sleep-Related Behaviours: Retrospective Questionnaire Assessment

These questionnaires each provide a unique perspective on the nature of the sleep disturbance. They were selected for inclusion because of their relevance to the behavioural component of the cognitive-behavioural model of insomnia. They included measures of sleep quality and perceived impairment, and the extent to which the participant engaged in behaviours that are incompatible with sleep.

Pittsburgh Sleep Quality Index (PSQI). This 19-item, self-report measure of general sleep quality assessed sleep as a multi-factorial phenomenon. It was designed for use in clinical populations (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989; see Appendix M). The PSQI has been shown to possess high specificity and sensitivity in distinguishing good and poor sleepers in medical and psychiatric populations (Buysse et al., 1989). The PSQI is retrospective in nature (respondents are asked to report for the past month). In the present study, the PSQI was used to assess habitual sleep onset latency (SOL), sleep duration, time spent in bed, sleep efficiency, sleep quality, sleep medication usage, and daytime dysfunction experienced within the past month. It also provided a global, summative score of subjective sleep impairment (range: 0 to 21, with high scores indicating worse sleep). The PSQI has been shown to have good internal consistency (Cronbach's alpha = 0.83; Buysse et al., 1989) and test-retest reliability ($r = 0.85$ for a

period of one month; Buysse et al., 1989) among patients with and without clinical diagnoses. The reliability and validity of the PSQI have also been established among medical populations (e.g., bone marrow transplant patients, breast cancer patients, and women with benign breast problems; Carpenter & Andrykowski, 1998). Moreover, the PSQI has also been used in studies of insomnia among chronic pain patients (e.g., Smith et al., 2000; Wilson et al., 1998), where it has been shown to be sensitive to treatment effects (e.g., Currie et al., 2000). However, the internal consistency for the PSQI may be somewhat lower in samples of all-insomniac patients. For example, Currie et al. (2000) reported a Cronbach's alpha of 0.64 among a group of individuals diagnosed with insomnia secondary to chronic pain. In the present study, the reliability coefficient for the global subscale was comparable to that reported by Currie et al. (2000), at 0.67. Nevertheless, the PSQI has become a standard measure in insomnia research, where it has been used to differentiate good from poor sleepers (Buysse et al., 1989).

Sleep Hygiene Usual Practice Scale. Adapted from the Sleep Hygiene Awareness and Practice Scale (Lacks, 1987; Lacks & Rotert, 1986; see Appendix N), this 19-item questionnaire comprises examples of sleep hygiene practices that can interfere with sleep (e.g., napping during the day, drinking more than 3oz. of alcohol before bedtime, exercising strenuously within 2 hours of bedtime). In addition, it includes items relating to sleep disturbance due to worry, noise, light, temperature, and bed partner. Respondents indicated the number of days or nights, on average, that they engage in these behaviours each week. The summed scores range from 0 to 133, with higher scores indicating increasingly poor sleep hygiene practices. Lacks' original scale has been shown to discriminate good from poor sleepers. Specifically, although poor sleepers have been

shown to possess greater knowledge of proper sleep hygiene practices, they tend to practise good sleep hygiene less frequently (Lacks & Rotert, 1986). The measure may only be marginally reliable, however; a Cronbach's alpha value of 0.53 was observed in the current study.

Sleep and Sleep-Related Behaviours: Daily Diary Measures

In addition to the interviews and written assessments, participants completed formal sleep diaries over the course of four nights.

Daily Sleep Diary (DSD). The sleep diary was based on a format described by Haythornthwaite, Hegel, et al., 1991 (see Appendix O). It assessed total sleep time (number of hours slept), time spent in bed, subjective sleep onset latency (estimated number of minutes it took to fall asleep), frequency of nocturnal awakenings, and sleep efficiency. In addition, it included ratings of sleep quality and a sense of feeling rested upon awakening. Although sleep data for insomniacs are inherently variable, this diary has been shown to possess adequate test-retest stability when recorded over a period of at least four days (i.e., Spearman-Brown reliability coefficients ranging from 0.69 to 0.87 for different sleep parameters; Haythornthwaite, Hegel, et al., 1991). Cronbach's alpha values over the four days of the present study were fair to good: 0.88 for sleep onset latency, 0.73 for wake episodes after sleep onset, 0.81 for sleep duration, 0.85 for sleep efficiency, and 0.67 for sleep quality.

Sleep Hygiene Daily Diary. This measure was completed each morning, and its contents were identical to the abovementioned Sleep Hygiene Usual Practice Scale. It was also based upon the work of Lacks (1987; Lacks & Rotert, 1986), but addressed daily, rather than usual, practice. It listed 19 examples of problematic sleep hygiene

behaviours. However, in the prospective portion of the study, respondents were asked to indicate whether or not they had engaged in any of the problematic sleep-related behaviours during the previous day (see Appendix P). One point was given for each behaviour that was endorsed, thus yielding a theoretical score of 0 to 19, with higher scores indicating more problematic sleep-related behaviours. An additional item, which asked participants to note the duration of any naps they had taken the previous day, was also added in the present study. The total scores for this new scale showed good internal consistency ($\alpha = 0.77$).

Sleep-Related Cognitions: Retrospective Questionnaire Assessment

Consistent with the cognitive-behavioural model of insomnia, the cognitive aspects of insomnia were assessed in addition to the behavioural aspects. The cognitive aspects included measures of perceived impairment due to insomnia, dysfunctional attitudes and beliefs about sleep, and general knowledge about sleep hygiene.

Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS). The DBAS (Morin, 1993; see Appendix Q) was selected as a global measure of problematic sleep-related cognitions. It is composed of 30 statements reflecting attitudes and beliefs regarding sleep that are commonly observed among individuals whose sleep is disturbed. Issues such as misconceptions about the causes of insomnia, misattributions or amplification of the consequences of sleep loss, unrealistic expectations regarding sleep, low perceptions of control over the sleep problem, and faulty beliefs about sleep-promoting practices are assessed in the DBAS. According to Morin (1993), faulty beliefs about sleep or the effects of sleep loss can lead to increased worry and arousal at night, and thereby lead to a more severe experience of insomnia. In its original version, respondents were asked to

indicate the extent of their agreement with each statement on a 10-cm. visual analogue scale with poles labeled 'strongly disagree' to 'strongly agree.' This format has been modified slightly for the present study; instead, a 10-point Likert scale was used (Means, Lichstein, Epperson, & Johnson, 2000). The total score of the DBAS has been shown to have adequate internal consistency among outpatient insomniacs, with Cronbach's alphas ranging from 0.72 (Espie, Inglis, Harvey, & Tessier, 2000) to 0.82 (Edinger et al., 2000) for the full scale. The current study yielded a Cronbach's alpha value of 0.88.

Sleep Hygiene Knowledge. Also adapted from Lacks' Sleep Hygiene Awareness and Practice Scale, this 50-item questionnaire (Lacks, 1987; Lacks & Rotert, 1986; see Appendix R) was developed to assess general awareness and application of sleep-related behaviours. This measure was used to assess general sleep hygiene knowledge, asking respondents to rate, on a seven-point scale (1 'very beneficial to sleep' to 7 'very disruptive to sleep'), the extent to which they perceive 13 specific behaviours (e.g., going to bed hungry, waking up at the same time each day) to affect sleep. Lacks' scoring protocol was employed: correct answers were given one point; incorrect answers three points, and omitted responses two points. This section thus has a range of 13 to 39, with higher scores indicating lower degrees of good knowledge of sleep hygiene practices. A Cronbach's alpha value of 0.64 was obtained in the present study.

Caffeine Knowledge. Another subscale of the Sleep Hygiene Awareness and Practice Scale (Lacks, 1987; Lacks & Rotert, 1986; see Appendix S), this measure was used to assess patients' general knowledge of caffeine-containing products. Participants were provided with a list of 18 common foods, beverages, and nonprescription medications (e.g., chocolate cake, regular tea, Dexatrim diet pills), and were asked to indicate whether

or not the item contains caffeine or any other stimulant. The caffeine knowledge score was derived by taking the number of items answered correctly and dividing them by the number of items attempted (respondents were instructed to omit any items they are not familiar with), thus yielding a theoretical score of 0 to 100% (Lacks, 1987; Lacks & Rotert, 1986).

Sleep-Related Cognitions: Daily Diary Assessment

Pre-Sleep Arousal Scale (PSAS). This 16-item questionnaire was chosen to assess arousal associated with cognitive and somatic anxiety. It is composed of two, eight-item subscales measuring cognitive and somatic hyperarousal specifically within the pre-sleep context (Nicassio et al., 1985; see Appendix T). Respondents are asked to indicate on Likert-type scales ranging from 1 ('not at all') to 5 ('extremely') the degree to which they experienced symptoms of anxious cognitive and somatic arousal while trying to fall asleep the night before. Symptoms such as increased heart rate, muscle tightness, and stomach upset are examples of somatic arousal; symptoms of cognitive arousal include racing thoughts, worrying, rumination, and high distractability. Each subscale has a possible range of eight to 40, and the total score a possible range of 16 to 80, with high scores indicating higher levels of arousal. Both subscales have been shown to possess adequate internal consistency (Cronbach's alphas = .76 to .88 among college students and insomniacs; Nicassio et al., 1985) and test-retest reliability ($r = 0.72$ for the cognitive subscale and $r = 0.76$ for the somatic subscale; Nicassio et al., 1985). Alpha values in the present study were excellent (0.92 for the somatic dimension, and 0.97 for the cognitive and combined dimensions). Subscale scores on the PSAS have been shown in earlier

research to distinguish good from poor sleepers and to correlate moderately with self-reported measures of sleep onset latency (Nicassio et al., 1985).

Mood-Related Measures: Retrospective Questionnaire Assessment

Beck Depression Inventory-II (BDI-II). The BDI-II (Beck, Steer, & Brown, 1996) is a 21-item self-report measure of depressive symptomatology experienced within the past two weeks. Respondents rated each symptom on a scale of 0-3; thus, summary scores could range from 0 to 63, with higher scores indicative of higher levels of depression. The BDI-II has been shown to possess high internal consistency among university samples ($\alpha = .93$) and psychiatric outpatients ($\alpha = .92$), and adequate content validity has been established (Beck et al., 1996). As discussed earlier, although chronic pain and depression may present with common symptoms (e.g., reduced concentration, insomnia, fatigue), recent research has shown that somatic symptoms retain their validity among samples of chronic pain patients. Furthermore, the BDI-II remains widely used in clinical practice with the medically ill (Arnau, Meagher, Norris, & Bramson, 2001). The BDI-II was shown to be highly reliable in the present analysis ($\alpha = 0.88$).

Mood-Related Measures: Prospective Daily Diary Assessment

Daily Mood Log. The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988; see Appendix U) comprised ten positive and ten negative adjectives describing emotional states. The positive and negative subscales are conceptualized as orthogonal to one another; that is, positive and negative affect were conceptualized as independent dimensions rather than opposite poles of the same dimension. Indeed, Clark et al. have shown that the PANAS subscales are not

significantly correlated. For the PANAS, respondents were asked to evaluate, on a 5-point scale, the extent to which each adjective was consistent with their current emotional state (1 = 'very slightly or not at all' to 5 = 'very much'), resulting in a possible range of 0 to 50 for each dimension. Clark and colleagues reported good internal consistency reliabilities for both positive and negative subscales (alphas = 0.90 and 0.87, respectively) in their student sample for ratings of the current day. Clark et al. also found that the scales were reliable for psychiatric inpatients (alphas = 0.85 and 0.91 for the positive and negative subscales, respectively), and had adequate test-retest reliability (0.79 and 0.81). In the present study, participants completed the PANAS in the morning and in the evening; excellent reliability was also established (alphas = 0.98 and .97 for positive and negative affect, respectively).

Group Classification

Participants were classified according to the following groups: chronic pain patients with major depressive disorder and chronic pain patients who did not meet the diagnostic criteria for major depression. Ideally, the current study would have subdivided participants into groups of depressed pain patients with and without insomnia and nondepressed participants with and without insomnia. However, inadequate numbers of patients belonging to all four categories were identified (i.e., very few depressed patients with chronic pain do not have insomnia). This is consistent with other research with this population (Wilson et al., 2002).

Data Analyses

The data were analyzed using SPSS, Version 13.0, for Windows. Initial screening was conducted to identify outliers with scores greater than +/- 3 standard deviations from

the mean. Very few outliers were found; they consisted of four depressed and two nondepressed individuals who reported extremely long napping episodes during the study period. The values for napping minutes for these participants were recoded as one minute longer than the next-highest score for napping within the respective group. The next-highest scores in each case fell within three standard deviations of the mean for that group.

The primary analyses were evaluated using a series of t -tests conducted within conceptual families of measures (Dar, Serlin, & Omer, 1994). Specifically, the measures and data were categorized into the following families of hypotheses: (1) pain and disability; (2) sleep and sleep-related behaviours; (3) sleep-related cognitions; (4) mood-related variables. Alpha-level corrections for multiple comparisons were conducted using Benjamini and Hochberg's (1995) method of controlling the false discovery rate. Thus, a familywise critical alpha level of .05 was applied to each theoretical group of hypotheses. The effects within a given family of hypotheses were then evaluated against a unique critical alpha value. According to this method, the ordinal placement of the size of the effect for a given variable within its respective hypothesis is divided by the number of variables within that hypothesis. This figure is then multiplied by the critical familywise alpha value. For instance, if a hypothetical group comprised 5 variables, then the largest effect was tested against a critical value equivalent to $(1/5)(.05) = .01$. Then, the second largest effect within that hypothetical family was tested against a critical value of $(2/5)(.05) = .02$, and so on. Benjamini and Hochberg (1995) note that these corrections are less conservative than the Bonferroni method, but permit some degree of control over the Type I error rate. For the exploratory hypotheses regarding the correlates of

interviewer-rated global insomnia severity and mood, the critical alpha levels were applied across the same theoretically-related families of hypotheses (e.g., pain and disability, sleep and sleep-related behaviours, sleep-related cognitions, and mood).

In order to ensure that the assumptions of the *t*-test were met, skewness and kurtosis values were computed for the variables of interest within each group. These values were adequate, with values generally falling within the 0 to +/- 1.50 range. For nondepressed individuals, SOL on the PSQI and the DSD were somewhat skewed, with values of 2.02 and 2.38, respectively. In addition, total time spent napping was positively skewed (2.99). Six variables were kurtotic: PSQI SOL (4.19), DSD SOL (6.35), PSQI WASO (2.53), napping time (8.44), and negative affect (2.22). In the depressed group, time spent napping was somewhat kurtotic (-2.09), as was global insomnia severity (3.83). However, a decision was made not to transform these variables, because the *t*-test is relatively robust to minor violations of assumptions and because so few variables were potentially problematic. In addition, Levene's test was employed in order to assess for the assumption of homogeneity of variance between the groups; none of the variances differed significantly (all *ps* > .05).

There were very little missing data. Two participants chose not to complete the Caffeine Knowledge scale; another did not complete the Sleep Hygiene Usual Practice measure. Two additional participants did not complete the PANAS. The remainder of missing data was observed within the daily diaries. In those cases, averages were computed based upon the data that were available (e.g., averages were computed over three days instead of four). However, this was necessary in only a handful of cases (*n* = 4). In instances where this was not possible, as in questionnaire data, the variables were

coded as 'missing' and the adjusted sample sizes and degrees of freedom for each statistical test have been noted.

Pre- and post-chronic pain treatment. A series of t -tests and χ^2 analyses was performed in order to evaluate differences between patients who had ($n = 12$) and had not ($n = 48$) previously completed treatment for chronic pain. No significant differences were observed between pre- and post-treatment groups regarding a diagnosis of major depression [$\chi^2(1) = .15, p = .70$] or insomnia [$\chi^2(1) = 1.98, p = .16$]. T -tests revealed that pre-treatment patients scored higher than their post-treatment counterparts with regard to global insomnia severity [$t(58) = 2.42, p = .019$], daily diary pain severity [$t(58) = 2.08, p = .042$], and total nap minutes on the daily dairies [$t(58) = 1.46, p = .017$]. However, once the Benjamini and Hochberg (1995) criteria for controlling familywise error rate were applied to each hypothetical family of hypotheses, these differences were not statistically significant. Thus, participants pre- and post-chronic pain treatment were combined in the analyses.

Results

Participant Characteristics

The initial sample consisted of 49 females and 17 males. One male and five female participants withdrew from the study following the interview and did not complete the questionnaire or diary assessments. These individuals were excluded from the data analysis; thus, the final sample consisted of 44 females (73.3%) and 16 males (26.7%). The average age of the participants was 46 years ($sd = 9.2$; range = 21 to 60 years). The preferred language for the majority of participants was English ($n = 54$), while one preferred French, and five patients were comfortable in either language. Fifty-six indicated they were of Caucasian ethnicity, two were Asian, and two described their ethnicity as "Other."

As presented in Table 6, participants with and without major depression did not differ significantly in terms of age, sex, time elapsed since the development of the chronic pain condition, or medication usage, measured by the MQS. The groups did not differ significantly regarding level of education or whether or not they were employed at the time of the study. Twenty participants (33.3%) were currently involved in some form of legal proceedings related to their pain condition at the time of the interview; again, this did not differ significantly between the groups.

The participants had suffered from pain for an average of 7 years ($sd = 5.6$; range = 9 months to 24.5 years). The primary location of the pain was in the lower back or lumbar spine area (35%), followed by pain throughout the entire body (23.3%), the neck area (16.7%), the shoulders (8.3%), and the legs and feet (6.7%). The remainder (8.3%) stated that their pain was located in the joints, pelvis, or arms and hands. Twenty-six

Table 6. Demographic Characteristics of Participants With and Without Major Depressive Disorder (MDD)

Variable	No MDD	MDD	Test Statistic	
	(<u>n</u> = 27)	(<u>n</u> = 33)	(<u>df</u>)	<u>p</u>
Age: <u>M</u> (<u>sd</u>)	46.93 (9.79)	45.24 (8.76)	<u>t</u> (58) = .70	.49
Sex (<u>n</u> , %)	17 Females (63)	27 Females (82)		
	10 Males (37)	6 Males (18)	χ^2 (1) = 2.7	.10
Time since chronic pain began (years): <u>M</u> (<u>sd</u>)	7.19 (6.71)	6.97 (4.63)	<u>t</u> (58) = .15	.88
Education: <u>n</u> (%)			χ^2 (1) = 1.8	.18
No Post-Secondary	11 (40.7)	13 (39.4)		
Any Post-Secondary	16 (59.3)	20 (60.6)		
Employment: <u>n</u> (%)			χ^2 (1) = .01	.92
No Employment	19 (70.4)	28 (84.8)		
Any Employment or Student	8 (29.6)	5 (15.2)		
Legal Involvement: <u>n</u> (%)	10 (37.04)	10 (30.30)	χ^2 (1) = .30	.58
Medication Usage: <u>M</u> (<u>sd</u>)	24.55 (15.93)	24.14 (10.95)	<u>t</u> (54) = .12	.91

participants (43.3%) indicated that their pain had begun spontaneously, while 14 (23.3%) had experienced motor-vehicle accidents, and 10 (16.7%) had been involved in work-related accidents. The remainder ($n = 9$, or 15%) had experienced some other form of accident, or the pain had begun following an illness ($n = 1$, or 1.7%).

Clinical Characteristics of Participants With and Without Major Depressive Disorder

The final sample consisted of 33 individuals who met DSM-IV criteria for major depression based on the PRIME-MD interviews, and 27 individuals who did not meet the diagnostic criteria. Thus, the rate of major depressive disorder in the present study was 55%. Of the 60 interviews, 14 (23.3%) were reviewed by a second rater with experience with structured diagnostic interviews; interrater agreement for the diagnosis of major depression was perfect ($\kappa = 1.00$, $p < .001$).

There were a number of clinical differences between the groups (see Table 7). For example, patients with major depression were significantly more likely to have a concurrent diagnosis of generalized anxiety disorder. Patients without major depression were more likely to have been diagnosed with major depression in partial remission. This is not surprising, given that the diagnoses of current major depression and major depression in partial remission are mutually exclusive. In addition, the groups did not differ regarding a history of major depression before the development of chronic pain.

Nevertheless, it is important to note that the group that did not receive a diagnosis of current major depressive disorder was not necessarily free of psychopathology. As shown in Table 7, 15 of the 27 participants (55.6%) without major depression qualified for a current diagnosis of another mental disorder.

Table 7. Comorbid Psychiatric Diagnoses for Participants With and Without Major Depressive Disorder (MDD)

Diagnosis	No MDD (n = 27)	MDD (n = 33)	χ^2 (1) (p)
Mental Disorders (n, %):			
Panic Disorder	3 (11.11)	10 (30.30)	3.22 (.07)
Generalized Anxiety Disorder	4 (14.81)	19 (57.58)	11.49 (.001)*
Major Depression In Partial Remission	7 (25.93)	0	9.69 (.002)*
Dysthymia	2 (7.41)	4 (12.12)	.37 (.55)
Bipolar Affective Disorder	0	3 (9.09)	2.58 (.11)
Pre-Pain History of Depression	14 (51.86)	17 (51.51)	.001 (.98)
Alcoholism (Current or Past)	2 (7.41)	1 (3.03)	.60 (.44)
Sleep Disorders (n, %):			
Insomnia	21 (77.78)	32 (96.97)	5.31 (.021)
Hypersomnia	5 (18.51)	15 (45.45)	4.85 (.028)
Sleep Apnea	4 (14.81)	6 (18.18)	.12 (.73)
Sleep Phase Disorder	2 (7.41)	1 (3.03)	.60 (.44)
Restless Legs Syndrome	5 (18.51)	6 (18.18)	.001 (.97)
Periodic Limb Movement Disorder	3 (11.11)	5 (15.15)	.21 (.65)
Nightmare Disorder	3 (11.11)	3 (9.09)	.074 (.80)
Sleep Terror Disorder	1 (3.7)	3 (9.09)	.69 (.41)
Sleepwalking Disorder	0	1 (3.03)	.83 (.36)

* Denotes statistical significance when holding error rate constant at .05 for each category of diagnosis (Benjamini & Hochberg, 1995).

Sleep Disorders

Interrater agreement on 14 of the 60 cases (23.3%) was perfect ($\kappa = 1.00$, $p < .001$) with regard to insomnia diagnosis. Insomnia was a very common problem among both groups of participants, with fully 53 out of 60 (88.3%) fulfilling DSM-IV criteria (see Table 7). Given the uniformly high prevalence, the rates of insomnia did not differ significantly between those with and without major depression when conservative statistical criteria were applied. A trend was observed for more participants with major depression to report clinically significant hypersomnia ($p = .028$), although again this did not achieve statistical significance. Eight individuals with major depression and four without reported having sleep difficulties before they developed chronic pain; this difference was not statistically significant.

Both groups reported similar rates of sleep apnea, restless legs syndrome, sleep phase disorder, and periodic limb movement disorder (with the qualification that definitive diagnoses of these disorders was not possible without PSG). Regarding parasomnias (nightmare disorder, sleep terror disorder, sleepwalking disorder), few differences were observed. No participants reported symptoms consistent with narcolepsy, dyssomnia NOS, or parasomnia NOS.

Even though no specific sleep disorders differed reliably between the groups, when 'any' sleep disorder was considered, the prevalence was higher among those with major depression. In total, 33 of the 53 patients with insomnia were also diagnosed with another dyssomnia or parasomnia. Of these, 12 did not have major depression, while 21 did [$\chi^2(1) = 21.08$, $p < .001$]. Of the 33 participants with insomnia and another sleep disorder, 18 also had hypersomnia, 15 of whom also had major depression. One

participant had hypersomnia but no current insomnia; this participant also had major depression. The remainder of the observed sleep disorders that co-occurred with insomnia or one other sleep disorder were as follows: probable obstructive sleep apnea ($n = 7$), circadian rhythm disorder ($n = 3$), restless legs syndrome ($n = 10$), periodic limb movement disorder ($n = 8$), nightmare disorder ($n = 6$), sleep terror disorder ($n = 3$), and sleepwalking disorder ($n = 1$).

In summary, with the exception of generalized anxiety disorder, both groups were approximately equal in terms of psychiatric comorbidity, although patients with major depression were significantly more likely to report insomnia with another comorbid sleep disorder. Nonsignificant trends toward patients with major depression having a greater prevalence of insomnia ($p = .021$) and hypersomnia ($p = .028$) were also observed.

Testing of Hypotheses

In general, for the main variables of interest, the majority was in the expected direction, and show that patients with major depression reported greater difficulty in functioning than patients without major depression on most measures of pain, disability, mood, and some, but not all, aspects of sleep.

The independent variables were grouped according to families of hypotheses: pain- and disability-related variables, sleep and sleep-related behaviours, sleep-related cognitions, and mood-related variables. As indicated earlier, Benjamini and Hochberg's (1995) method of controlling for Type-I error rate was then applied to each family of hypotheses ($\alpha = .05$, two-tailed).

Hypothesis 1: Pain and Disability

The means, standard deviations, effect sizes, confidence intervals, and *post-hoc* power analyses are presented in Table 8. Patients with major depression reported significantly higher levels of pain on all but one of the measures. Specifically, they reported significantly higher levels of total pain, current pain, least pain, and average pain on retrospective reports. Indeed, the only retrospective pain-related measure that did not differentiate the groups was the rating of the worst pain experienced in the past two weeks. For participants in both groups, these ratings were uniformly high. Data derived from the daily diary pain severity ratings also showed significantly higher levels of pain among participants with major depression. Moreover, patients with major depression reported significantly higher levels of disability than those without major depression. Medium to large effect sizes ($d_s = .59$ to $.89$) and statistical power (64% to 94%) were achieved among all of the pain ratings, with the exception of retrospective estimates of worst pain.

Hypothesis 2: Sleep and Sleep-Related Behaviours

Self-reported sleep parameters. As indicated earlier, the groups did not differ significantly with regard to the DSM-IV diagnosis of insomnia, at least when conservative statistical criteria were applied. Nevertheless, the results were in the hypothesized direction (see Table 9), and perhaps indicate a trend ($p = .021$), for a greater proportion of participants with major depression to receive a diagnosis of insomnia than those without major depression. Clinically significant insomnia was clearly very prevalent in both groups, however. The ratings of insomnia severity were also high for both groups (i.e., 4.48 to 5.06 out of a maximum rating of 6). Again, there was a trend

Table 8. Pain and Disability Measures for Participants With and Without Major Depression (Hypothesis 1)

Variable	No MDD (n = 27)		MDD (n = 33)		t(df)	p	d (Power, %)	95% Confidence Interval of the Difference	
	Mean (sd)	Mean (sd)	Lower	Upper					
Retrospective Pain Ratings (past 2 weeks):									
Current Pain	5.81 (1.76)	7.15 (1.84)	-2.86 (58)	.006*	.75 (82)	-2.27	-2.27	-1.10	-1.10
Worst Pain	8.67 (1.39)	9.12 (1.11)	-1.41 (58)	.16	.36 (28)	-2.71	-2.71	-2.05	-2.05
Least Pain	3.59 (2.13)	5.03 (2.68)	-2.26 (58)	.027*	.60 (64)	-2.05	-2.05	-1.27	-1.27
Average Pain	5.93 (1.52)	7.12 (1.73)	-2.81 (58)	.007*	.73 (81)	-2.29	-2.29	-1.27	-1.27
Total Pain	24.00 (5.41)	28.42 (6.56)	-2.81 (58)	.007*	.73 (82)	-2.29	-2.29	-1.27	-1.27
Diary Pain Ratings	5.83 (1.53)	7.28 (1.70)	-3.42 (58)	.001*	.89 (94)	-2.29	-2.29	-1.27	-1.27
Disability Score	26.00 (8.52)	31.15 (9.01)	-2.26 (58)	.028*	.59 (62)	-9.72	-9.72	-5.6	-5.6

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995).

Table 9. Sleep and Sleep-Related Behaviours for Participants With and Without Major Depressive Disorder (MDD; Hypothesis 2)

Variable	No MDD (n = 27)		MDD (n = 33)		Test Statistic (df)	p	d (Power, %)	95% Confidence Interval of the Difference	
	Mean (sd)	Mean (sd)	Lower	Upper					
Global Insomnia	4.48 (1.05)	5.06 (1.00)	t (58) = -2.18	.03	.57 (59)	-1.11	-0.05		
Insomnia - Past Week	4.48 (1.09)	4.97 (1.13)	t (58) = -1.69	.10	.44 (40)	-1.07	.09		
PSQI:									
SOL (mins)	51.11 (49.62)	67.91 (61.07)	t (58) = -1.51	.25	.30 (22)	-46.00	12.41		
WASO (mins)	149.04 (91.13)	151.21 (132.60)	t (53) = -.07	.94	.02 (5)	-61.48	57.14		
Sleep Efficiency	63.52 (16.88)	62.52 (19.81)	t (57) = .21	.84	.05 (6)	-8.70	10.70		
Sleep Duration (hrs)	5.35 (1.38)	5.23 (1.80)	t (58) = .28	.78	.07 (6)	-.73	.96		
Global Insomnia	14.48 (4.06)	15.48 (3.68)	t (58) = -1.00	.32	.26 (17)	-3.01	1.00		
Sleep Hygiene Usual Practice	28.31 (13.80)	35.61 (15.30)	t (57) = -1.90	.06	.50 (49)	-15.00	.40		

(cont'd)

Table 9 (cont'd). Sleep and Sleep-Related Behaviours for Participants With and Without Major Depressive Disorder (MDD: Hypothesis 2)

Variable	No MDD		MDD		Test Statistic	p	d (Power, %)	95 % Confidence Interval of the Difference	
	Mean (sd)	(n = 27)	Mean (sd)	(n = 33)				Lower	Upper
DSD:									
SOL (mins)	43.76 (37.02)		52.94 (49.98)		t (58) = -.79	.43	.03 (13)	-32.37	14.01
WASO (mins)	125.45 (91.01)		131.90 (88.67)		t (57) = -.27	.78	.07 (6)	-53.41	40.52
Sleep Efficiency	69.40 (16.39)		65.46 (17.97)		t (57) = .87	.39	.23 (14)	-5.10	12.97
Sleep Duration (hrs)	6.12 (1.48)		5.78 (1.81)		t (58) = .80	.43	.21 (13)	-.52	1.21
Sleep Quality	4.30 (1.62)		3.93 (1.71)		t (58) = .86	.39	.22 (14)	-.49	1.24
Any Naps: n (%)	15.00 (55.56)		18.00 (54.54)		$\chi^2 (1) = .61$.44	.20 (5)	-.36	1.62
Total Nap Minutes:									
M (sd)	95.85 (206.48)		159.18 (256.77)		t (58) = -1.04	.30	.27 (19)	-185.69	59.03
Sleep Hygiene Diary	13.7 (5.28)		18.94 (7.52)		t (58) = -3.05	.003*	.81 (88)	-8.67	-1.80

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995).

for those with major depression to receive higher global ratings of insomnia severity ($p = .03$). Medium effect sizes ($d_s = .44$ and $.57$) were observed for retrospective measures of self-reported insomnia severity (global and past week), although neither dimension achieved statistical significance when conservative statistical criteria were applied.

Although the groups did not differ significantly on specific sleep parameters on the PSQI or daily diaries, it is important to note that both groups reported substantially disturbed sleep. For instance, participants without major depression reported an average SOL of approximately 44 and 50 minutes on the DSD and the PSQI. These individuals also reported an average of over two hours of time awake after sleep onset on the PSQI and the DSD. This group reported very low rates of sleep efficiency on both the PSQI and the daily diary reports (63.5% and 69.4%, respectively), well below the rate of 85% generally used to distinguish good from poor sleepers (Lacks & Morin, 1992). They also reported very few hours of sleep per night (5.35 on the PSQI and 6.12 on the DSD, on average).

Similarly, the sleep parameters of individuals with major depression were also problematic. These participants reported an average SOL of approximately 53 minutes on the DSD, and 68 minutes on the PSQI. They too estimated an average of over two hours of time awake after sleep onset on the PSQI and the DSD. Average sleep efficiency was also very low on both retrospective and daily diary reports (approximately 62.5% on the PSQI and 65.5% on the DSD), as was sleep duration (approximately five hours on the PSQI and six hours on the DSD). Thus, although data on sleep parameters did not differ significantly between the groups, this was related to the fact that both groups evidenced substantial sleep impairment. This is further buttressed by the fact that both groups rated

their sleep quality as an average of approximately four on a scale of one to ten (with a score of ten reflecting excellent sleep quality) over the course of the study.

Sleep-related behaviours. Even though the self-reports of these traditional sleep parameters did not differ between the groups, other aspects of sleep-related behaviours did differ between participants with and without major depression. Specifically, individuals with major depression were significantly more likely to report poor sleep-related habits on the daily diary measures ($p = .003$; $d = .81$, $1 - \beta = .88$). A medium effect size ($d = .50$) was also observed for the retrospective ratings of poor sleep hygiene, although the comparison did not achieve statistical significance ($p = .06$).

Hypothesis 3: Sleep-Related Cognitions

Table 10 presents the values obtained for sleep-related cognitions. As hypothesized, participants with major depression reported significantly higher levels of dysfunctional attitudes and beliefs about sleep, as measured by the DBAS ($p = .025$), with a medium effect size ($d = .60$). Excellent statistical power and a large effect size were obtained for the caffeine knowledge scale, with participants with major depression evidencing significantly less understanding about caffeine-containing products ($p = .007$; $d = .74$, $1 - \beta = .82$).

The groups did not differ in their general sleep hygiene knowledge; indeed, this dimension yielded an extremely low effect size and statistical power. The results suggesting roughly equal sleep hygiene knowledge between groups is potentially noteworthy, given that depressed patients reported engaging in significantly poorer sleep hygiene behaviours in the daily diary portion of the study.

Table 10. Sleep-Related Cognitions for Participants With and Without Major Depressive Disorder (MDD; Hypothesis 3)

Variable	No MDD		MDD		t(df)	p	d (Power, %)	95% Confidence	
	Mean (sd)	n	Mean (sd)	n				Lower	Upper
		27		33				Interval of the Difference	
DBAS	137.52 (33.23)		159.82 (40.25)		-2.31 (58)	.025*	.60 (65)	-41.66	-2.94
Sleep Hygiene Knowledge	20.78 (5.20)		21.24 (4.78)		-.36 (58)	.72	.09 (6)	-3.05	2.12
Caffeine Knowledge	76.45 (10.19)		67.86 (12.74)		2.79 (56)	.007*	.74 (82)	2.42	14.76
PSAS Totals:									
Cognitive Arousal	65.74 (21.20)		84.42 (30.62)		-2.69 (58)	.009*	.71 (80)	-32.61	-4.76
Somatic Arousal	56.48 (17.06)		65.42 (20.46)		-1.81 (58)	.07	.47 (46)	-18.82	.93
Total Arousal	122.22 (34.93)		149.85 (45.66)		-2.58 (58)	.012*	.68 (76)	-49.03	-6.23

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995).

In terms of anxious mood assessed specifically within the presleep context, patients with major depression reported significantly higher levels of anxious cognitive and total arousal (p s = .009 and .012, respectively), with moderately large effect sizes (d s = .71 and .68, respectively) and acceptable statistical power (80% and 76%, respectively). A nonsignificant trend was observed for anxious somatic arousal (p = .07), in addition to a medium effect size (d = .47).

Exploratory Analyses: Mood-Related Variables

Not surprisingly, participants with major depression scored significantly higher on the BDI-II (see Table 11), although even those participants without a formal diagnosis of major depression had scores that were quite elevated relative to the general population. The participants with major depression were also significantly more likely to endorse items on the PANAS reflecting negative affect over the course of the four days of the study (p < .001). Excellent statistical power (99% for negative affect and 100% for the BDI-II) and effect sizes (d s = 1.13 and 1.76, respectively) were observed among these indices; however, virtually no effect and extremely low statistical power were observed for positive affect.

Exploratory Analyses: Correlates of Global Insomnia Severity

The correlates of global insomnia severity, as assessed by the interviewer rating, were also examined. Because patients with major depression have traditionally been excluded from this type of correlational investigation (e.g., Currie et al., 2000; see Smith & Perlis, 2006, and Setpanski & Rybarczyk, 2006, for recent reviews), we examined the pattern of correlations separately for those diagnosed with and without major depression.

Table 11. Mood-Related Variables for Participants With and Without Major Depressive Disorder (MDD; Exploratory Hypothesis)

Variable	No MDD (<i>n</i> = 27)		MDD (<i>n</i> = 33)		<i>t</i> (<i>df</i>)	<i>p</i>	<i>d</i> (Power, %)	95% Confidence Interval of the Difference	
	Mean (<i>sd</i>)	Mean (<i>sd</i>)	Lower	Upper					
BDI-II	20.41 (7.85)	34.58 (8.55)	-6.62(58)	<.001*	1.76 (100)	-18.45	-9.89		
PANAS Totals:									
Positive Affect	145.42 (41.62)	148.03 (61.18)	-.19(56)	.85	.05 (5)	-30.82	25.61		
Negative Affect	112.65 (38.36)	165.84 (54.52)	-4.20(56)	<.001*	1.13 (99)	-78.57	-27.81		

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995).

Interrater reliability for global ratings of insomnia was high (intraclass $r = .79$, $p < .001$). Several trends were observed within and between the two depression groups in terms of global insomnia severity for participants with and without major depressive disorder (see Table 12). For individuals without major depression, interviewer-rated reports of global insomnia severity were correlated significantly with most measures of pain, and all indices of presleep arousal. Global insomnia severity, as rated by the interviewer, was also correlated significantly with retrospective reports of poor sleep-related behaviours. Nonsignificant trends were observed between global insomnia severity and PSQI global insomnia, sleep duration, and sleep efficiency.

For individuals with major depression, all measures of pain and disability were significantly associated with global insomnia severity. However, global insomnia severity was also related to many of the retrospective and prospective indices of sleep on the PSQI and the DSD. Specifically, global insomnia severity was significantly associated with increased SOL and global insomnia on the PSQI, in addition to decreased sleep efficiency. On the DSD, global insomnia severity was significantly related to decreased sleep efficiency and sleep quality. There was also a tendency for sleep duration to be negatively correlated with global insomnia severity, although this was not significant when conservative statistical criteria were applied. There were also nonsignificant trends for insomnia severity to be related to presleep somatic and total arousal on the PSAS among participants with major depression.

A series of Fisher's z -transformations was performed in order to compare statistically the correlation coefficients for each group. None of the correlation coefficients differed significantly between the groups. Thus, although the pattern of the

Table 12. Pearson and Point-Biserial Correlation Coefficients for Global Insomnia Severity (Exploratory Hypothesis)

Variable	No MDD				MDD				Fisher's z
	95% Confidence Interval		95% Confidence Interval		95% Confidence Interval		95% Confidence Interval		
	r (p)	Lower	Upper	d (Power, %)	r (p)	Lower	Upper	d (Power, %)	
Current Pain	.09 (.65)	-.03	.45	.18 (7)	.57 (<.001)*	.28	.76	1.39 (96)	.04
Worst Pain	.43 (.025)*	.06	.70	.95 (62)	.59 (<.001)*	.31	.78	1.46 (97)	.45
Least Pain	.43 (.024)*	.06	.70	.95 (62)	.44 (.01)*	.11	.68	.98 (75)	.96
Average Pain	.53 (.005)*	.19	.76	1.25 (85)	.47 (.006)*	.15	.70	1.07 (82)	.76
Total Pain	.46 (.016)*	.10	.72	1.04 (70)	.56 (.001)*	.27	.76	1.35 (95)	.60
Diary Pain Severity	.49 (.009)*	.14	.73	1.12 (77)	.63 (<.001)*	.37	.80	1.62 (99)	.47
Disability Subscale	.20 (.32)	-.19	.54	.41 (16)	.47 (.006)*	.15	.70	1.07 (82)	.25

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995). (cont'd)

Table 12 (cont'd). Pearson and Point-Biserial Correlation Coefficients for Global Insomnia Severity (Exploratory Hypothesis)

Variable	No MDD (n = 27)			MDD (n = 33)			Fisher's z	
	r (p)	Lower	Upper	r (p)	Lower	Upper		
		95% Confidence Interval			95% Confidence Interval			
			d (Power, %)			d (Power, %)		
Sleep and Sleep-Related Behaviours:								
Insomnia Diagnosis	.08 (.70)	-.31	.45	.16 (7)	-.33	.35	.02 (5)	.81
Nap (mins.)	.26 (.20)	-.13	.58	.54 (24)	-.55	.10	.52 (28)	.04
PSQI:								
SOL	.34 (.09)	-.05	.64	.72 (40)	.15	.70	1.07 (82)	.56
WASO (mins.)	.25 (.23)	-.14	.56	.52 (22)	.01	.62	.75 (46)	.70
Sleep Duration	-.48 (.012)	-.73	-.12	1.09 (75)	-.65	-.07	.87 (65)	.73
Sleep Efficiency	-.39 (.04)	-.67	-.01	.85 (52)	-.69	-.13	1.01 (76)	.81
Global Insomnia	.49 (.009)	.14	.73	1.12 (77)	.38	.81	1.67 (99)	.44

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995). (cont'd)

Table 12 (cont'd). Pearson and Point-Biserial Correlation Coefficients for Global Insomnia Severity (Exploratory Hypothesis)

Variable	No MDD				MDD				Fisher's z
	r (p)		95% Confidence Interval		r (p)		95% Confidence Interval		
	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper	
SOL	.29 (.14)	-.10	.61	.61 (30)	.27 (.13)	-.08	.56	.56 (32)	.94
Sleep Duration	-.22 (.28)	-.55	.17	.45 (18)	-.29 (.11)	-.56	.06	.30 (36)	.78
Sleep Efficiency	-.27 (.17)	-.59	.12	.56 (26)	-.45 (.011)*	-.69	-.13	1.01 (76)	.96
Sleep Quality	-.04 (.85)	-.41	.35	.08 (5)	-.50 (.003)*	-.72	-.19	1.15 (88)	.06
Sleep Hygiene Usual Practice	.62 (.001)*	.31	.81	1.58 (96)	.24 (.19)	-.11	.54	.49 (26)	.08
Sleep Hygiene Diary Practice	.18 (.37)	-.21	.52	.37 (13)	.20 (.26)	-.15	.51	.41 (19)	.94

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995). (cont'd)

Table 12 (cont'd). Pearson and Point-Biserial Correlation Coefficients for Global Insomnia Severity (Exploratory Hypothesis)

Variable	No MDD				MDD				Fisher's z	
	r (p)		95% Confidence Interval		r (p)		95% Confidence Interval			
	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper		
Sleep-Related Cognitions:										
DBAS	.30 (.13)	.61	-.09	.10 (.32)	.23 (.19)	.53	-.12	.47 (.24)	.76	
Sleep Hygiene Knowledge	.06 (.78)	.43	-.33	.12 (.6)	.23 (.21)	.53	-.12	.47 (.24)	.61	
Caffeine Knowledge	-.13 (.54)	.26	-.49	.26 (.9)	.19 (.31)	.50	-.16	.39 (.17)	.27	
PSAS:										
Cognitive Arousal	.49 (.009)*	.73	.14	1.12 (.77)	.19 (.29)	.50	-.16	.39 (.17)	.20	
Somatic Arousal	.51 (.007)*	.75	.16	1.19 (.81)	.44 (.01)	.68	.11	.98 (.75)	.75	
Total Arousal	.55 (.003)*	.77	.22	1.32 (.89)	.32 (.07)	.60	-.03	.68 (.44)	.31	

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995). (cont'd)

Table 12 (cont'd). Pearson and Point-Biserial Correlation Coefficients for Global Insomnia Severity (Exploratory Hypothesis)

Variable	No MDD (n = 27)		MDD (n = 33)		Fisher's z
	r (p)	95% Confidence Interval Lower Upper d (Power, %)	r (p)	95% Confidence Interval Lower Upper d (Power, %)	
Mood-Related Variables:					
BDI-II	.25 (.21)	-.14 .56 .52 (23)	.22 (.23)	-.13 .52 .45 (22)	.90
PANAS:					
Positive Affect	.06 (.76)	-.33 .43 .12 (6)	.09 (.63)	-.26 .42 .18 (7)	.93
Negative Affect	.11 (.60)	-.28 .47 .22 (8)	.21 (.26)	-.14 .52 .43 (19)	.72

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995).

associations differed somewhat between the groups, none of the correlation coefficients for retrospective self-reports of global insomnia severity differed significantly between the groups after controlling for familywise error rate. In general, then, the overall pattern of correlations was largely comparable between the groups.

Exploratory Analyses: The Correlates of Major Depression, Depression Symptoms, and the Targets of CBT for Insomnia

One purpose of the present study was to evaluate whether or not it might be appropriate to consider CBT for insomnia among patients with major depression. Thus, it should be noted that, across the various families of hypotheses, the targets of CBT for insomnia that are regarded as amenable to treatment, such as dysfunctional attitudes and beliefs about sleep, poor sleep hygiene practices, daytime napping, and presleep arousal, were associated with depressive symptoms. In fact, when these variables were treated as a hypothetical family unto themselves, and a slightly modified critical level for statistical significance was applied to reflect a different number of variables in the conceptual family (Benjamini & Hochberg, 1995), the majority were significantly correlated with BDI-II scores (see Table 13).

Specifically, depression severity was significantly correlated with prospective and retrospective measures of sleep-related behaviours, as reflected by scores on the Sleep Hygiene Daily Practice and Sleep Hygiene Daily Practice scales. Additionally, dysfunctional attitudes and beliefs about sleep, and all indices of presleep arousal were significantly associated with elevated depression severity. There was also a nonsignificant trend for BDI-II scores to be negatively associated with caffeine knowledge.

Table 13. Pearson and Point-Biserial Correlation Coefficients for Depression Severity, Major Depressive Disorder (MDD), and the Targets of Cognitive-Behavioural Therapy for Insomnia (Exploratory Hypothesis)

Variable	BDI-II		95% Confidence Interval		MDD		95% Confidence Interval	
	r (p)	Lower	Upper	d (Power, %)	r (p)	Lower	Upper	d (Power, %)
Sleep Hygiene Knowledge	.006 (.97)	-.25	.26	.01 (5)	.05 (.72)	-.21	.30	.10 (7)
Caffeine Knowledge	-.27 (.04)	-.49	-.02	.56 (54)	-.35 (.007)*	-.55	-.11	.75 (78)
Sleep Hygiene Usual Practice	.30 (.021)*	.05	.51	.31 (64)	.24 (.06)	-.01	.47	.50 (45)
Sleep Hygiene Daily Practice	.42 (.001)*	.19	.61	.93 (93)	.36 (.005)*	.12	.56	.77 (82)
Napping	.13 (.31)	-.13	.37	.26 (16)	.14 (.30)	-.12	.38	.29 (18)
DBAS	.50 (<.001)*	.28	.67	1.15 (99)	.29 (.025)*	.04	.51	.61 (62)
PSAS Cognitive Arousal	.41 (.001)*	.18	.60	.90 (92)	.33 (.009)*	.08	.54	.70 (74)
PSAS Somatic Arousal	.36 (.005)*	.12	.56	.77 (82)	.23 (.07)	-.03	.46	.47 (42)
PSAS Total Arousal	.43 (.001)*	.20	.62	.95 (95)	.32 (.012)*	.07	.53	.68 (71)

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995).

In order to further evaluate the applicability of CBT for insomnia among individuals with major depression, the point-biserial correlation coefficients were also examined for the presence or absence major depression as a dichotomous diagnosis and the traditional targets for change in CBT for insomnia. As presented in Table 13, statistically significant point-biserial correlations were observed between major depression and low levels of caffeine knowledge, daily diary reports of sleep hygiene, and dysfunctional attitudes and beliefs about sleep ($r = .29, p = .025$). Presleep cognitive and total anxiety were also significantly associated with a diagnosis of major depression.

Partial correlation coefficients. Because the magnitude of the correlations observed between pain severity and insomnia were more robust than those observed between depression and insomnia, and because significant differences were observed between the groups on pain-related variables, a decision was made to investigate the correlates of depression while controlling for pain severity. Partial correlation coefficients were computed for major depression and BDI-II scores with the targets of CBT for insomnia, with prospective pain severity ratings held constant. As presented in Table 14, many of the associations were rendered nonsignificant with this adjustment, with the exception of the DBAS, and presleep cognitive and total arousal, which remained significantly correlated with BDI-II scores.

Table 14. Partial Correlation Coefficients for Depression Severity, Major Depressive Disorder (MDD), and the Targets of Cognitive-Behavioural Therapy for Insomnia, Controlling for Daily Diary Pain Severity (Exploratory Hypothesis)

Variable	BDI-II			95% Confidence Interval			MDD			95% Confidence Interval		
	r	(p)		Lower	Upper	d (Power, %)	r	(p)		Lower	Upper	d (Power, %)
Sleep Hygiene Knowledge	-.004	(.98)		-.26	.25	.01 (5)	.07	(.61)		-.19	.32	.14 (8)
Caffeine Knowledge	-.21	(.13)		-.44	.05	.43 (36)	-.31	(.023)		-.52	.06	.65 (68)
Sleep Hygiene Usual Practice	.23	(.10)		-.03	.46	.47 (42)	.14	(.30)		-.12	.38	.28 (18)
Sleep Hygiene Daily Practice	.30	(.028)		.05	.51	.63 (65)	.21	(.12)		-.05	.44	.43 (36)
Napping	-.018	(.98)		-.27	.24	.04 (5)	0	(1.00)		-.25	.25	0
DBAS	.45	(.001)*		.22	.63	1.01 (96)	.18	(.18)		-.08	.41	.37 (27)
PSAS Cognitive Arousal	.33	(.015)*		.08	.54	.70 (74)	.25	(.07)		-.004	.47	.52 (49)
PSAS Somatic Arousal	.29	(.033)		.04	.51	.61 (62)	.08	(.57)		-.18	.33	.16 (9)
PSAS Total Arousal	.35	(.01)*		.11	.55	.75 (80)	.21	(.13)		-.05	.44	.43 (36)

* Denotes statistical significance when holding familywise error rate constant at .05 for each hypothesis (Benjamini & Hochberg, 1995).

Discussion

The current study sought to compare the sleep characteristics of chronic pain patients who either did or did not present with current major depressive disorder. Although this question has been addressed before in the chronic pain literature, the available studies to date have used global measures of insomnia severity applied retrospectively to participants' recollections of 'typical' sleep. The present study was able to replicate this methodology, and extend it by incorporating a concurrent daily diary approach to the assessment of sleep, pain, and mood. Moreover, the present study incorporated a higher standard of diagnostic assessment by using standardized structured interviews as well as questionnaires. The selection of questionnaires was guided by current conceptualizations of the psychology of insomnia, which may permit some speculation about the cognitive and behavioural factors relevant to the maintenance of insomnia in depressed individuals with chronic pain. Ultimately, the role of CBT in alleviating the insomnia of chronic pain and depression will require clarification in clinical trials. The present findings, however, highlight some of the specific issues that may require particular attention in this regard.

Most studies to date on the nonpharmacological intervention for insomnia have excluded participants with comorbid major depression; however, a small body of literature (e.g., Morin et al., 1990) has suggested that CBT for insomnia within this patient group may indeed be appropriate (see Stepanski & Rybarczyk, 2006, for a review). Insomnia in chronic pain and/or major depression is, arguably, a secondary phenomenon. In the current study, ten participants (16.7%) had a history of insomnia before they developed chronic pain, but, as with other studies of chronic pain patients, the vast majority report that their sleep problems were either caused, or significantly

exacerbated, by the development of chronic pain (Morin et al., 1998). Regardless of its precipitating cause, it is thought that insomnia can be perpetuated by specific cognitive and behavioural processes. These perpetuating factors are thought to turn an acute phase of insomnia into a chronic state (Morin, 1993; see Figure 2). Thus, intervention at the level of these perpetuating factors, regardless of the initial cause of the insomnia, might have the potential to improve sleep among individuals with or without comorbid major depression. Nevertheless, some aspects of CBT for insomnia (e.g., sleep restriction) may exacerbate the depressive state (Smith & Perlis, 2006). Therefore, the current study sought to identify the specific precipitating factors, from a cognitive-behavioural perspective, before clinical trials are attempted within this vulnerable patient population (Smith & Perlis, 2006; Stepanski & Rybarczyk, 2006).

Participant Characteristics

Comorbid mental disorders. Participants with and without current major depressive disorder displayed the same rates of other mental disorders, with one notable exception. Individuals with major depression reported more comorbid generalized anxiety disorder than individuals without major depression. This may be due to the fact that generalized anxiety and major depression share many diagnostic features (APA, 1994). Indeed, most relevant epidemiological studies of comorbidity have reported that the two syndromes are found commonly among the same individuals (Kessler, DuPont, Berglund, & Wittchen, 1999; Stein & Heimberg, 2004). It may also be related to the fact that participants with major depression experienced more pain, and were thus more worried, agitated, and distressed. It is also conceivable that the anxious state of patients with GAD leads to

increased pain-related worry and increased perceptions of pain severity, thus making the individual more prone to major depression.

Sleep disorders. Insomnia was a common problem in this sample, experienced by most participants in both groups. This is generally in keeping with many studies documenting sleep disturbance among individuals with chronic pain (see Table 3), although the finding that fully 53 of the 60 participants (88.3%) fulfilled the DSM-IV criteria for insomnia disorder is a higher prevalence than has been found in most other research. This is perhaps attributable to the nature of the treatment program for chronic pain that is offered at The Rehabilitation Centre. This program is directed toward helping individuals cope with a chronic pain problem that has been resistant to treatment with other modalities of care. Therefore, the sample may be skewed by the nature of the referral base -- patients who, by definition, are having difficulty coping with pain and its psychological consequences.

The high prevalence of insomnia among the participants without major depression was unexpected to some extent. It is important to note, however, that insomnia is a diagnostically significant symptom for many other mental disorders than fully syndromal major depression, including major depression in partial remission and generalized anxiety disorder. These diagnoses were both documented among participants without current major depression.

Hypothesis 1: Pain and Disability

Clear differences were observed between the depressed and nondepressed groups on measures of pain and disability, with individuals with major depression exhibiting more severe problems. This is consistent with a large body of previous research (Benjamin et

al., 1988; Currie & Wang, 2004; Elliott et al., 2003; Haley et al., 1985; Haythornthwaite, Sieber, et al., 1991), and may be due to the proposed bidirectional relationships between pain and mood (Chiu et al., 2005; Gureje et al., 2001), whereby one problem accentuates the consequences of the other. For instance, epidemiological data have suggested that the presence of an anxiety or depressive disorder at baseline was a significant predictor of chronic pain 12 to 24 months later (Gureje et al., 2001; Currie & Wang, 2005). Similarly, Feldman et al. (1999) conducted time-lagged within-subject analyses from daily diaries collected from chronic pain patients over 28 days. They found that daily pain ratings predicted depressed mood the subsequent day, and conversely, depressed mood predicted subsequent pain ratings. In terms of psychological intervention, it may therefore be that the successful treatment of pain may improve mood, and vice versa. For instance, many recent treatment studies investigating CBT interventions for pain have shown significant improvements in mood as well (Doncel, Vaz Leal, De Peralta, Vazquez, & Macias, 2004; Evers et al., 2002; Jensen et al., 2001; Spinhoven et al., 2004). In a similar vein, the proposed bidirectional relationships between pain and mood suggest that CBT for depression may ameliorate pain severity; future research should seek to evaluate whether or not this is the case.

Hypothesis 2: Sleep and Sleep-Related Behaviours

Surprisingly, few significant differences were observed between the groups on actual indices of sleep, such as sleep onset latency, time awake after sleep onset, sleep efficiency, and sleep duration. This may be due to a ceiling effect, consistent with the fact that both groups reported high rates of insomnia and other sleep disorders. That is,

chronic pain itself may be so disruptive of sleep that the concurrent experience of major depression does not introduce further problems in a strictly additive way.

It should be noted, however, that the current study relied solely on self-reports of sleep, which, some have argued (e.g., Menefee et al., 2000), may compromise the validity of sleep parameter estimates. Certainly, the PSG literature has found differences between depressed and nondepressed patients on various aspects of sleep architecture. This was not assessed in the present study, so we are not able to comment on the electrophysiological aspects of sleep.

In terms of sleep-related behaviours, no differences were observed regarding retrospectively reported 'typical' behaviours; this is inconsistent with the work of Woodley and Smith (2006), who found that individuals with high levels of depressive symptoms reported significantly more sleep-disruptive behaviours than those with low levels of depression symptoms. However, in the current study, participants with major depression nevertheless reported poorer sleep-related practices during the daily diary portion of the study. These hypotheses are supported by the fact that the groups did not differ significantly in terms of general sleep hygiene knowledge. Thus, although it appears that both groups are aware of what to do to improve sleep, and both groups reported roughly similar 'typical' sleep hygiene practices, patients with major depression did not practise those behaviours as consistently as patients without major depression. This is similar to the findings of Lacks and Rotert (1986), and Harvey (2000), who found that poor sleepers were less likely to engage in sleep-compatible behaviours, despite the fact that they possessed similar (Harvey, 2000) or even higher levels (Lacks and Rotert, 1986) of such knowledge. Woodley and Smith (2006), however, did find that participants

with high levels of depressive symptoms reported significantly more sleep-incompatible behaviours and beliefs than those with fewer symptoms.

The current study extends these findings to individuals with major depression. Persons with major depression, by definition, tend to have low motivation (APA, 1994), and this may be why they fail to put what they appear to know into practice. Taken together, the findings regarding typical sleep hygiene reports, actual sleep hygiene, and general sleep hygiene knowledge suggest that behavioural interventions, particularly those targeted toward relapse prevention and increased adherence to sound sleep hygiene practices (Riedel & Lichstein, 2001), may require a greater emphasis among patients with major depression. Furthermore, these results lend support for the utility of the adapted daily version of the Sleep Hygiene Practice Scale (Lacks, 1987; Lacks & Rotert, 1986), which was created specifically for use in the present study.

Hypothesis 3: Sleep-Related Cognitions

Although the groups did not differ significantly in terms of general sleep hygiene knowledge, participants with major depression scored lower than their nondepressed counterparts in the area of caffeine knowledge. Perhaps information regarding the ubiquity of caffeine-containing products has not permeated common sleep hygiene knowledge. However, this does not explain why low levels of caffeine knowledge are associated with major depression. It may be that individuals with little caffeine knowledge tend to inadvertently consume more caffeine, which can contribute to insomnia, and insomnia can often be a precursor to depression (Breslau et al., 1996; Chang et al., 1997; Ford & Kamerow, 1989; Roberts et al., 2000). Furthermore, the daytime fatigue typically associated with both depression and insomnia may lead to

increased intentional caffeine consumption (Lacks & Rotert, 1986), thus perpetuating the cycle of poor sleep and adverse daytime consequences. However, these proposed linkages between major depression and caffeine knowledge are speculative at best, and more research would be necessary in order to elucidate these relationships. Nonetheless, these findings suggest that education regarding the ubiquity of caffeine-containing products may be particularly useful in treating patients with major depression.

The current study also provides support for the utility of addressing erroneous attitudes and beliefs about sleep when treating patients with major depression and insomnia. Many studies have highlighted the efficacy of interventions such as cognitive restructuring in addressing dysfunctional attitudes and beliefs about sleep in the treatment of primary insomnia (e.g., Morin et al., 1993, 1994, 2002). However, the current study provides support for the notion that these attitudes are also held by individuals with comorbid major depression, and thus represent a valid target for insomnia intervention within this population. The fact that depressed individuals reported significantly higher levels of dysfunctional attitudes may be a reflection of an underlying attitude structure that is, by definition, generally negative among individuals with major depression (Beck, 1976; 2005). It may also be that these dysfunctional attitudes and beliefs contribute to insomnia, and therefore to depression, in light of the research highlighting the interdependency of the phenomena. For instance, large-scale longitudinal data suggest that insomnia can be a prodromal symptom of depression (Breslau et al., 1996; Chang et al., 1997; Ford & Kamerow, 1989; Mallon et al., 2000; Ohayon & Roth, 2003; Perlis, Giles, Buysse, Tu et al., 1997; see Fava, 2004; Thase, 2005; Riemann & Voderholzer,

2003, for reviews). At the very least, the present findings suggest some communality of cognitive, attitudinal processes between major depression and insomnia.

There was also a tendency for individuals with major depression to report higher levels of anxious cognitive and somatic arousal in the presleep period. This is not surprising, given that arousal-reducing interventions, such as relaxation training, are well-documented as stand-alone treatments for insomnia (Means et al., 2000; Morin et al., 1999). In addition to its positive impact upon sleep, relaxation training has also been shown to bring about improvements in dysfunctional attitudes and beliefs about sleep (Means et al., 2000). According to Morin (1993), presleep emotional arousal, and its associated insomnia, are the natural consequences of maladaptive attitudes and beliefs about sleep. Therefore, cognitive restructuring may also have an important role to play in helping patients reduce their levels of preseleep arousal.

It is possible that increased levels of arousal may also be due to the comorbidity between major depression and generalized anxiety observed within the present study. Belanger, Morin, Gendron, and Blais (2005) found that presleep cognitive arousal was elevated in insomniacs, particularly among those with GAD. Similarly, Harvey (2001) found that the presleep cognitive content of insomniacs was more focused on worries, problems, and noises than that of noninsomniacs.

Exploratory Hypotheses: Mood-Related Variables and the Correlates of Insomnia

Severity

Not surprisingly, participants with and without major depression differed significantly on depression severity and negative affect. This served to further underscore the validity of the diagnoses of major depression.

In terms of the interviewer's global ratings of insomnia severity, the most robust associations were observed with pain severity ratings among participants with major depression. In general, however, the correlates of insomnia severity did not differ significantly between the groups. Although this is relatively circumstantial, rather than definitive evidence, it lends support to the suggestion that similar insomnia treatments (i.e., CBT) may benefit chronic pain patients with comorbid major depression, given that the therapeutic efficacy of CBT for insomnia among chronic pain patients without major depression has been documented in the literature (Currie et al., 2000; Edinger et al., 2006). That is, because the correlation coefficients are broadly comparable, it argues for an underlying similarity in the sleep problems experienced by chronic pain patients with and without major depressive disorder. Those with major depression may be functioning at a lower level than those without, but the pattern of correlations is similar.

Exploratory Hypothesis: Major Depression, Depression Severity, and the Targets of CBT for Insomnia

When the correlational analyses were focused specifically on the targets of CBT interventions (which reduced the number of statistical comparisons to which corrections were applied), the majority were indeed significantly associated with depressive symptoms. A diagnosis of major depression was associated with several of these treatment targets as well, albeit to a lesser extent. This may be due to the inherently lower degree of variability when calculating correlation coefficients with a dichotomous variable, such as the presence or absence of a diagnosed disorder, rather than an ordinal measure, such as the BDI-II. In the latter case, the broader distribution of possible scores allows for a finer degree of concordance between variables to emerge.

Although the correlations between depression scores and the target variables of CBT for insomnia add further support to the suggestion that the treatment may be applicable to patients with major depression, this conclusion must be tempered by a consideration of the role of pain.

Importantly, the partial correlations between major depression, depression symptoms, and the targets of CBT for insomnia suggest that pain severity may play the most significant role in the emergence of insomnia, even among participants with major depression. Specifically, most significant correlations were rendered nonsignificant when pain severity was partialled out. The exception in this regard was that, even when pain severity was held constant, dysfunctional attitudes and beliefs about sleep still retained a significant association with depression. These results are contrary to those reported by Smith et al. (2000), who found that presleep arousal was more highly associated with sleep complaints than pain severity; however, Smith et al.'s (2000) sample did not consider major depressive disorder, and their assessments were purely retrospective in nature. Nonsignificant trends were also observed for other targets of CBT for insomnia (e.g., presleep arousal, daily sleep hygiene practices). For most measures, however, the partialing out of the contribution of pain severity greatly diminished the correlations between depression measures and the targets of CBT for insomnia. This suggests that treatments for insomnia within this population might benefit from a close integration of pain and sleep interventions; indeed, such interventions might well have positive effects on mood as well (Backhaus et al., 2001; Evers et al., 2002; Jensen et al., 2001; Spinhoven et al., 2004). However, in practical terms, and given the strong emphasis placed upon physical conditioning, psychoeducation, and pain-specific CBT in pain management

programs, there may be the potential to 'overload' patients when too many interventions are applied at the same time. In this context, therefore, it may be advisable to introduce the sleep interventions after patients have undergone pain management, and have received instruction in the general cognitive-behavioural model and have a better understanding of pain self-management. As some investigators have noted, patients with chronic pain tend to attribute their sleep disturbance entirely to the experience of pain (Morin et al., 1998; Nicassio & Wallston, 1992; Wilson et al., 2001). Therefore, without some prior focus on the issue of pain, there may be some potential for patients to be skeptical of the possible benefit of an insomnia intervention that does not focus directly upon pain relief.

Limitations of the Current Study and Suggestions for Future Research

Several methodological issues warrant mention when considering the interpretation of the results of the current study. The large number of correlational analyses highlight the obvious difficulties in attributing cause and effect relationships to the variables. Additionally, although short-term longitudinal data were collected in the present study, the analyses were cross-sectional in nature. Nevertheless, data collection of this kind is valuable in that it provided multiple data points and measured behaviours within the context in which they occurred. Indeed, Jensen and McFarland (1993) have shown that multiple ratings (i.e., at least 12) of pain severity over several days result in estimates of average pain ratings that have excellent psychometric properties (i.e., internal consistency, test-retest stability, and validity coefficients of 0.90 or more) within a sample of chronic pain patients. In fact, the daily diary reports of sleep, sleep-related thoughts and behaviours, and mood collected in the present study showed good internal

consistency and argue for the utility of assessing these factors *in situ* over time, rather than retrospectively based upon memory and impression.

Another drawback of the current study was the large proportion of women (73.3%). However, given that chronic pain is more common in women, this is not surprising (Eriksen et al., 2004; Saastamoinen et al., 2005). The relatively low participation rate among patients who were approached about the study (17.6 %) was another limitation. Due to the voluntary nature of research with human participants, studies of this kind are often limited in the number of participants that can be recruited within a reasonable period of time. Indeed, patients referred to multi-disciplinary pain management programs have typically exhausted all other medical avenues and are experiencing significant difficulties in functioning. Given that these individuals are quite incapacitated and are so frequently in contact with the health care system, the relatively low participation rate observed in the present study is perhaps not surprising. In fact, participation rates of this magnitude are common in other research projects conducted at The Rehabilitation Centre (Currie et al., 2000). Future research of this kind would benefit from the knowledge that a relatively long period of recruitment may be necessary to accrue large samples for complex prospective designs. Additionally, voluntary participation by nature carries with it the possibility that individuals who agree to participate may not be representative of the broader population. For instance, the most incapacitated individuals might have chosen not to participate because of the extra effort involved. Conversely, higher-functioning individuals may have not been interested in participating because they did not feel the study was personally relevant to them. Unfortunately, for ethical reasons, no data were available on patients who declined to participate.

Treatment Implications

The results of the present study support the potential utility of both cognitive and behavioural interventions for insomnia among patients with chronic pain and comorbid major depression, and provide an empirical rationale for further study. Despite the methodological limitations, there was clear evidence that patients with major depression have both cognitive (dysfunctional attitudes and beliefs about sleep, high presleep arousal, poor caffeine knowledge) and behavioural (poor sleep hygiene practices) characteristics that may well contribute to their sleep problem, to an even greater extent than with patients who do not have major depression. The sleep disturbance itself is not necessarily more severe than for patients without major depression, but the perpetuating factors may be more robust and entrenched.

The next logical step in this program of research would involve treatment-based studies comparing CBT interventions for insomnia among patients with and without major depression. In fact, insomnia interventions may complement pain- and mood-based interventions (Woodley & Smith, 2006). For instance, there is research to suggest that sleep interventions may also further aid patients be helpful in reducing pain severity (Currie et al., 2000) and depressive symptoms (Backhaus et al., 2001). This notion is further supported by research documenting that poor nighttime sleep is associated with increased pain the following day (Affleck et al., 1996; Nicassio et al., 2002).

Some advance recommendations may be appropriate in order to optimize these interventions. Although insomnia treatment is a valid goal in its own right, pain and mood management should perhaps be undertaken first, given the level of engagement required for such programs, and the fact that treatment of depression and improvement in

self-management of pain may also alleviate insomnia. The results of the current study also suggest that dysfunctional attitudes and beliefs about sleep and presleep arousal constitute valuable individual targets of intervention, irrespective of pain severity or mood disorder. Furthermore, some psychoeducational aspects of insomnia treatment (i.e., knowledge of caffeine-containing products) appear to warrant special attention, as do interventions emphasizing relaxation and adherence to behavioural practices (Riedel & Lichstein, 2001). It is noteworthy in this context that some widely disseminated treatment manuals for depression actually contain modules derived from CBT for insomnia (Bieling & Antony, 2003; Leahy & Holland, 2000; Paterson, 2002), with the assumption that this approach has merit for the treatment of depressed individuals.

Some aspects of CBT for insomnia, however, are best applied with caution to people with chronic pain and major depression (Smith & Perlis, 2006). For example, it may be deleterious for individuals with suicidal ideation to be instructed to stay up much later than their usual bedtime, even if their usual bedtime is too early. Sleep restriction may also temporarily exacerbate symptoms of fatigue or pain. The sleep restriction intervention, in particular, includes a dimension of sleep deprivation that could be disconcerting for people who are physically and emotionally fragile. Certainly, notifying patients ahead of time that these effects may occur and should be transient would be advisable. Such individuals would likely benefit from careful monitoring of their mood. In fact, it may be advantageous to exclude patients with current or past suicidal ideation from initial intervention studies, until the clinical utility of CBT for insomnia is further established in psychiatric populations.

In summary, insomnia is a very common problem for patients with chronic pain. Comorbid major depression, however, appears to add an additional burden to these individuals as well. Major depression among chronic pain patients appears to be associated with higher levels of pain and other adverse consequences. The results of the present study suggest that psychological interventions for insomnia may have the potential to improve the functioning of patients with chronic pain and major depression. Furthermore, these improvements may have implications that go beyond the realm of sleep and perhaps alleviate pain and mood symptoms as well. Indeed, in light of the degree of suffering associated with these disorders, and the evidence indicating the potential utility of CBT for insomnia within this population, it would be remiss to neglect this promising avenue of research.

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Appendix A¹

Information Sheet

- Project:** Major depression and insomnia among chronic pain patients:
A prospective study
- Investigators:** Patricia C. Emery, B.A.(Hons), Ph.D. candidate
Keith G. Wilson, Ph.D., C.Psych., Staff Psychologist, The Rehabilitation
Centre

We are conducting a study on sleep among people who have problems with chronic pain. The purpose of the study is to evaluate the quality of sleep among people with pain problems, and to identify factors that may be associated with good or poor sleep. These factors include emotional health, pain, sleep-related thoughts and habits, and dreams. Participation in the study involves an interview, the completion of several questionnaires, and the completion of 4 days of sleep, mood, pain, and dream diaries.

If you agree to take part in the study, you will be interviewed at the Rehabilitation Centre or at your home. If the interview is conducted at the Centre, you will be reimbursed for your parking expenses. The interview will take approximately one hour and will contain questions regarding your sleep and emotional health. Please note that this interview will be audiotaped. This is for research purposes only, to check the accuracy of the interviewer's ratings, and the tape will contain no identifying characteristics. We will also give you questionnaires that ask about your pain, sleep, and emotional health. In addition, you will be asked to complete a sleep, pain, and mood diary over 4 consecutive days and nights. Completion of these diaries will take approximately 15 minutes per day. These records, including the audiotaped interview, will be kept for a period of ten years, and then be erased or destroyed.

If you decide to participate in this study, we would also be interested in looking at your Rehabilitation Centre chart. This will help us to know about your medical condition and treatments that you have been receiving.

Some of the specific questions that will be answered by this study are: What proportion of people with chronic pain have trouble sleeping? Do people who have good or poor sleep differ from one another in any medical or psychological ways? Do they have different habits, attitudes, and behaviours around sleep? How is the quality of dreams affected by the experience of chronic pain? Your participation in this study would help to answer these questions.

¹ This Appendix was printed on paper bearing the letterhead of The Rehabilitation Centre.

You may not personally benefit from your participation in the study, but the information you provide may be helpful in planning future treatments for people whose sleep is disturbed by chronic pain.

Your participation in this study is voluntary. You may withdraw from this study at any time, and your treatment at The Rehabilitation Centre will not be affected in any way. Personal information collected as part of this study will be kept confidential and it will not be shared with anyone outside of the research team. The data will be kept in a locked cabinet, and no identifying information will appear on any reports or publications. Participation in the study involves a certain degree of self-disclosure, so you may experience some emotional discomfort as you are interviewed about your emotional health. If you experience any distress during the course of the study, please let the interviewer/researcher know, and feel free to discuss the matter with him or her. You may also choose to withdraw from the study at any time for this reason, or choose not to answer any individual questions in the interview, on the questionnaires, or in the diaries.

If you have any questions about this study, please contact Patricia Emery at The Rehabilitation Centre (737-7350, ext. 5317), or Dr. Shawn Marshall, Chairperson of the Research Ethics Board at The Rehabilitation Centre (737-7350, ext. 5590).

Appendix B**Major depression and insomnia among chronic pain patients:****A prospective study with ambulatory monitoring**

If you would be willing to participate and would like to know more about the study,
please leave your name and telephone number below:

Name (please print):

Telephone number:

THANK YOU

Appendix C²

Recruitment Letter to Former Patients

**Project: Major depression and insomnia among chronic pain patients:
A prospective study**

Investigators: Patricia C. Emery, B.A.(Hons), Ph.D. candidate,
University of Ottawa
Keith G. Wilson, Ph.D., C.Psych., Staff Psychologist, The
Rehabilitation Centre

Dear Sir or Madam,

The above researchers at the Rehabilitation Centre are conducting a study about sleep problems affecting patients who have attended the Pain Clinic at the Centre. We are conducting this study because many people with chronic pain report that their sleep is disturbed, but there is little research into this problem.

If you decide to participate in the study, you would be asked to complete an interview, complete several questionnaires, and keep diaries on your pain, sleep, emotional health, and dreams over the course of 4 days. The interview would be conducted at the Rehabilitation Centre or at your home. If you decide to have the interview conducted at the Centre, you would be reimbursed for your parking expenses or bus fare. The interview would take about an hour and would contain questions regarding your pain, sleep, emotional health, and dreams; it would also be audiotaped. This is for research purposes only and would contain no identifying characteristics (these tapes, along with the other records from the study, will be kept for a period of ten years and then be erased or destroyed). The questionnaires and diaries also contain questions about pain, sleep, and emotional health. Completion of the diaries would be expected to take approximately 15 minutes each day, over the course of 4 days.

Please be aware that your participation in this study would be completely voluntary. Any treatment you may still receive at the Clinic would in no way be affected should you decide or decline to participate in the study. You may also withdraw from the study at any time, and your treatment would not be affected. Any personal information collected during the study will be kept confidential and will not be shared with anyone outside of the research team (Ms. Emery and Dr. Wilson).

In conducting this research, we hope to learn more about the sleep-related attitudes and behaviours of people with chronic pain, with the ultimate goal of identifying proper treatment strategies.

² This letter was printed on paper bearing the letterhead of The Rehabilitation Centre

A member of the research team will be in touch with you in January, 2005, to ask if you may be interested in participating in this study. If you are certain at this time that you do *not* wish to participate, kindly call 737-7350, ext. 5317, and leave a message with your name and telephone number.

Sincerely,

Dr. Usha Buenger
Medical Director, Chronic Pain Service

Appendix D

Telephone Recruitment Script

Hello. My name is _____, and I'm calling from The Pain Clinic at The Rehabilitation Centre. I am a doctoral student in psychology, and am conducting a study here at the Centre. I'm calling to follow up on the sign-up sheet you returned to us at the Pain Clinic orientation session.

There are certain conditions, however, that would preclude you from participating in this study. Patients suffering from epilepsy or another seizure disorder, who have chronic obstructive pulmonary disorder, acquired brain injury, cancer, lupus, or heart disease, or are over the age of 60, or women who are pregnant, are unfortunately not eligible for this study. Do any of these conditions apply to you?

We are conducting a study on sleep, mood, and dreaming among patients with chronic pain. The purpose of the study is to evaluate the quality of sleep among persons with chronic pain, and to identify the factors that are associated with good or poor sleep. Participation in the study entails an interview, the completion of several questionnaires, and the completion of 4 days of sleep, pain, and mood diaries.

If you agree to take part in the study, you will be interviewed at the Rehabilitation Centre or at your home. If you come to The Centre for your interview, you will be reimbursed for your parking expenses. The interview will take approximately one hour and will contain questions regarding your sleep, and mood. The interview will also be audiotaped; this is for research purposes only and the tape will contain no identifying characteristics. We will also give you questionnaires that ask about your pain, sleep, and mood. In addition, you will be asked to complete a sleep, mood, and pain diaries over 4 consecutive days and nights. Completion of these diaries will take approximately 15 minutes per day. These records, including the audiotape, will be kept for a period of ten years, and then be erased or destroyed.

Your participation in this study is completely voluntary. You may withdraw from this study at any time, and your treatment at The Rehabilitation Centre will not be affected in any way. Personal information collected as part of this study will be kept confidential at it will not be shared with anyone outside of the research team. At a later date, we will ask your permission to share these records with the clinical team of the Rehabilitation Centre.

Do you have any questions about the study? Do you think you would be interested in participating in it?

Appendix E

DECLARATION OF INFORMED CONSENT

Major Depression and insomnia among chronic pain patients:

A prospective study

Patricia C. Emery, B.A.(Hons), Ph.D. candidate (737-7350, ext. 5317)

Keith G. Wilson, Ph.D., C.Psych., Staff Psychologist,
The Rehabilitation Centre (737-7350, ext. 5608)

I have read the Information Sheet and have been told about the purpose of the study. Any questions I have about the study have been answered. I understand that I will be taking part in an interview that will last approximately one hour, in addition to completing questionnaires and diaries about my pain, sleep, and emotional health over the course of 4 days. I understand that the interview will be audiotaped, and that this is for research purposes only and the tape and all other documentation will be kept completely confidential. I understand that the audiotaped interview and the other records from the study will be kept for a period of ten years, and then will be erased or destroyed. I understand that some parts of my participation may be somewhat stressful, and I know that I can withdraw my consent at any time and the assessment will be stopped. I also agree that the researchers working on this project can review my Rehabilitation Centre chart.

I agree to participate in this study with the understanding that information will be collected and used for research purposes only and will be treated as confidential. I have been informed about the purpose of this study and realize that I am under no obligation to participate and may withdraw at any time. Refusal to participate or withdrawing from the study will in no way affect my present and/or future treatment at The Rehabilitation Centre.

Participant: _____ Date: _____

Investigator: _____ Date: _____

If you would like to be provided with the results of this study, please write your full name and address in the spaces provided:

Name: _____

Address: _____

Appendix F

PATIENT QUESTIONNAIRE – PRIME-MD

NAME:

TODAY'S DATE: _____

Instructions: *This questionnaire will help us better understand problems that you may be having. You may be asked more questions about some of these items. Please make sure to check every item.*

During the PAST MONTH, have you been bothered A LOT by...	YES	NO
1. Little interest or pleasure in doing things?	___	___
2. Feeling down, depressed, or hopeless?	___	___
3. "Nerves" or feeling anxious or on edge?	___	___
4. Worrying about a lot of different things?	___	___
5. Have you had an anxiety attack (suddenly feeling fear or panic)?	___	___
6. Have you thought you should cut down on your drinking of alcohol?	___	___
7. Has anyone complained about your drinking?	___	___
8. Have you felt guilty or upset about your drinking?	___	___
9. Was there ever a single day in which you had five or more drinks of beer, wine, or liquor?	___	___

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Now I am going to ask you some questions about your emotional health.

PANIC DISORDER

1. *In the past month, have you ever had an anxiety attack where you had a sudden feeling or fear or panic?
(What was that like?)*
1 = YES
2 = NO → Go to Generalized Anxiety Disorder

2. *Has this ever happened before?*
1 = YES
2 = NO → Go to Generalized Anxiety Disorder

3. *Does the attack sometimes come suddenly out of the blue, for no apparent reason?
(Even in situations where you don't expect to be nervous or uncomfortable?)*
1 = YES
2 = NO

4. *Have you worried a lot about having another attack?*
1 = YES
2 = NO

5. *Think about your last really bad attack.*

***Interviewer:** Once 4 of the following symptoms have been coded as YES, go to Generalized Anxiety Disorder.

- | | | |
|---|---------|--------|
| <i>(a) Were you short of breath?</i> | 1 = YES | 2 = NO |
| <i>(b) Did your heart race, skip, or pound?</i> | 1 = YES | 2 = NO |
| <i>(c) Did you have chest pain or pressure?</i> | 1 = YES | 2 = NO |
| <i>(d) Did you sweat?</i> | 1 = YES | 2 = NO |
| <i>(e) Did you feel as if you were choking?</i> | 1 = YES | 2 = NO |
| <i>(f) Did you have hot flashes or chills?</i> | 1 = YES | 2 = NO |
| <i>(g) Did you have nausea or an upset stomach, or the feeling you were going to have diarrhea?</i> | 1 = YES | 2 = NO |
| <i>(h) Did you feel dizzy, unsteady, or faint?</i> | 1 = YES | 2 = NO |
| <i>(i) Did you have tingling or numbness in Parts of your body?</i> | 1 = YES | 2 = NO |
| <i>(j) Did you tremble or shake?</i> | 1 = YES | 2 = NO |
| <i>(k) Were you afraid you were dying?</i> | 1 = YES | 2 = NO |

GENERALIZED ANXIETY DISORDER**ANXIETY**

6. *Have you been feeling nervous, tense, or anxious?*

(How bad does it get?)

(Does it come and go?)

(Do you feel that way most of the time?)

(Is it a problem for you?)

(How much does it bother you?)

0 No Anxiety

1 Slight: e.g., only occasionally has feelings of anxiety at a low level; not regarded as a particular problem

2 Mild: e.g., sometimes feels somewhat anxious or nervous, but not excessively and not most of the time; occasionally regarded as a minor problem

3 Moderate: e.g., definite periods of uncomfortable anxiety; usually feels at least somewhat nervous or anxious; regarded as a significant problem.

4 Strong: e.g., most of the time feels uncomfortably anxious; anxiety is regarded as a prominent and ongoing problem.

5 Severe: e.g., almost all of the time feels anxious; regarded as a troubling, serious, and ongoing problem.

6 Extreme: e.g., constant, unrelieved feelings of severe anxiety; regarded as a pervasive, consuming, and constant problem.

***Interviewer:** if ANXIETY < 3, then go to MAJOR DEPRESSION

***If >= 3:**

7. *Do you feel this way every day, or nearly every day? (On more than half the days in the past month?)*

1 = YES

2 = NO

8. *In the past month, have you been bothered by any of these problems nearly every day?*

***Interviewer:** Once 3 of the following symptoms have been coded YES, go to Item #9.

(a) *Feeling restless so that it is hard to stay still?* 1 = YES 2 = NO

(b) *Getting tired very easily?* 1 = YES 2 = NO

(c) *Muscle tension, aches, or soreness?* 1 = YES 2 = NO

(d) *Trouble falling asleep or staying asleep?* 1 = YES 2 = NO

(e) *Trouble concentrating on such things as reading, or watching TV?* 1 = YES 2 = NO

(f) *Becoming easily annoyed or irritated?* 1 = YES 2 = NO

9. *In the past month, has your anxiety made it hard for you to work, take care of things, you wanted to do, or get along with people?*

1 = YES

2 = NO → go to MAJOR DEPRESSION

10. *In the last 6 months, have you been worrying a great deal about different things?
(What kind of things have you been worrying about?)*

1 = YES

2 = NO → go to MAJOR DEPRESSION

***Interviewer:** Code as YES only if also YES to “*Has this been on more than half the days in the last 6 months?*”

11. *When you are worrying this way do you find that you cannot stop?*

1 = YES

2 = NO

MAJOR DEPRESSION

DEPRESSION

12. *Have you been feeling ‘down,’ or depressed?*

(How bad does it get?)

(Does it come and go?)

(Do you feel that way most of the time?)

(Is that a problem for you?)

0 No Depression

1 Minimal: e.g., only occasionally has feelings of being ‘down’ or depressed at a low level; not regarded as a particular problem.

2 Mild: e.g., sometimes experiences periods of being ‘down’ or depressed, but not excessively and not most of the time; occasionally regarded as a minor problem.

3 Moderate: e.g., definite periods of feeling ‘down’ or depressed; usually feels at least somewhat depressed; regarded as a significant problem.

4 Strong: e.g., most of the time feels quite depressed; regarded as a prominent and ongoing problem

5 Severe: e.g., almost all of the time feels very depressed; depression is regarded as a troubling, serious, and ongoing problem

6 Extreme: e.g., constant, unrelieved feelings of severe depression; regarded as a pervasive, consuming, and constant problem.

If >= 2:

13. *Do you feel this way every day, or nearly every day?*

Nearly Every Day 1 = YES 2 = NO

14. *How long have you felt this way?*

(For more than 2 weeks?)

Duration \geq 2 weeks 1 = YES 2 = NO

LOSS OF INTEREST OR PLEASURE

15. *Do you find that you have little interest or pleasure in doing things?*

(How bad does it get?)

(Does it come and go?)

(Have you lost interest in almost all activities, or only a few?)

(Is it a problem for you?)

(I'm not thinking so much about your ability to do things as about your interest in them.)

(even activities that don't require much physical effort, like enjoying your family, friends, reading, or watching T.V.?)

0 No Loss of Interest or Pleasure

1 Minimal: e.g., only occasionally experiences loss of interest or pleasure at a low level; not regarded as a particular problem.

2 Mild: e.g., sometimes experiences a loss of interest or pleasure in some activities but retains interest in others; occasionally regarded as a minor problem.

3 Moderate: e.g., definite loss of interest or pleasure in many activities; regarded as a significant problem.

4 Strong: e.g., most of the time feels a markedly diminished interest or pleasure in almost all activities; regarded as a prominent and ongoing problem

5 Severe: e.g., almost all of the time feels no interest or pleasure in any activities; regarded as a troubling, serious, and ongoing problem

6 Extreme: e.g., complete inability to experience any interest or pleasure in any activities, virtually all the time; regarded as a pervasive, consuming, and constant problem.

If \geq 3:

16. *Do you feel this way every day or nearly every day?*

Nearly Every Day 1 = YES 2 = NO

17. *How long have you felt this way?*

(more than a couple of weeks?)

Duration \geq 2 weeks 1 = YES 2 = NO

***Interviewer:** if both DEPRESSION $<$ 2 and LOSS OF INTEREST OR PLEASURE $<$ 3, then go to ALCOHOL ABUSE/DEPENDENCE.

18. For the past 2 weeks, have you had any of the following problems nearly every day?

***Interviewer:** Give credit without asking for symptoms already acknowledged in the GENERALIZED ANXIETY DISORDER module.

(a) **SOCIAL WITHDRAWAL**

Have you had less to do with people than usual?

(Have you been avoiding people?)

(Have you preferred to be by yourself?)

(Have you turned down or avoided any situations where you knew you would be with people?)

(Have you stopped calling your friends or answering the telephone?)

1 = YES 2 = NO

(b) (i) **BROODING**

Have you been worrying a lot?

(How much do you worry?)

(What kinds of things have you been worrying about?)

(How much of your time is spent in this?) (Are you able to get your mind off it?)

1 = YES 2 = NO

***Interviewer:** Note behaviour in interview as well.

***OR:** (ii) **SELF-PITY**

***Interviewer:** Note behaviour in interview. Behaviour and remarks indicate self-indulgent focusing on his/her own sorrows, problems, or misfortunes. In judging the severity, note the extent to which he/she demonstrates the following: (1) suffering is directly communicated without restraint in order to elicit sympathy from others; (2) personal problems are viewed as unique or more severe than those suffered by others; and (3) feels that he/she is not being helped or understood by others.

1 = YES 2 = NO

***OR:** (iii) **PESSIMISM**

Have you been discouraged (pessimistic, felt hopeless)?

What kind of future so you see for yourself?

(How do you think things will work out?)

(Can you see yourself or your situation getting any better?)

1 = YES 2 = NO

***Interviewer:** Note behaviour in interview as well.

(c) **DEPRESSED APPEARANCE**

Would you say that you look depressed to others?

(How much of the time?)

(Do you neglect your personal appearance?)

(Has it been so severe that other people could notice?)

1 = YES 2 = NO

***Interviewer:** Note behaviour in interview as well.

(d) *Feeling bad about yourself – or that you are a failure or have let yourself or your family down?*

1 = YES 2 = NO

(e) LACK OF REACTIVITY TO PLEASANT EVENTS

During the week when you were feeling the worst, did feeling depressed ever go away when you got your mind on other things or when something pleasant happened – like talking to a friend, or hearing good news, or did you feel bad no matter what was happening? (If someone tried to cheer you up, could they?)

1 = YES 2 = NO

***If YES:** *Would you say that you do not feel much better, even temporarily, when something good happens?*

1 = YES 2 = NO

(e) *Being do fidgety or restless that you were moving around a lot more than usual?*

1 = YES 2 = NO

***If no:** *What about the opposite – moving or speaking so slowly that other people could have noticed?*

***Interviewer:** Code as YES if YES to either question or if psychomotor agitation or retardation is observed during interview.

(f) *Trouble falling or staying asleep, or sleeping too much?*

1 = YES 2 = NO

How about waking up too early?

1 = YES 2 = NO

***Interviewer:** Code as YES if waking up at least 2 hours before usual time of awakening.

(g) *Feeling tired or having little energy?*

1 = YES 2 = NO

(h) *Poor appetite or overeating?*

1 = YES 2 = NO

re: **POOR** appetite:

- 0 No information
- 1 Not at all – normal or **INCREASED**
- 2 Slight decrease of questionable clinical significance
- 3 Mild decrease
- 4 Moderate decrease
- 5 No appetite, but forces self to eat

6 No appetite, and had to be fed

***Interviewer:** Code as YES if > 3

(i) *Have you lost weight recently?*

1 = YES 2 = NO

How much? _____ lbs.

***Interviewer:** Code as YES of >= 10 lbs.

(j) *Trouble concentrating on things, such as reading the newspaper or watching television?*

(k) *Being so fidgety or restless that you were moving around a lot more than usual?*

1 = YES 2 = NO

***IF NO:** *What about the opposite – moving or speaking so slowly that other people could have noticed?*

(Code as YES if YES to either question or if psychomotor agitation or retardation is marked and observed during the interview).

(l) *In the last 2 weeks, have you had thoughts that you would be better off dead, or of hurting yourself in some way?*

1 = YES 2 = NO

***Interviewer:** If YES, ask “Tell me about it.”

Comments:

MELANCHOLIA

19. **DISTINCT QUALITY OF MOOD.** Extent to which the depressed feelings are felt by the subject to be qualitatively different from the kind of feeling he or she would have or has had following the death of a loved one (not just more severe, or mixed with other symptoms, such as loss of interest).

Is this feeling of [use the patient's terms] different from the usual feelings that you would get, or have had after someone close to you died? (or from a sad movie or story?)

(How is the feeling different?)

0 No information or unable to understand question

1 No difference or just more severe

- 2 Questionable or minimal difference
- 3 Definitely different, but only mildly so
- 4 Very different

***Interviewer:** Code as YES if > 2

20. **DIURNAL MOOD VARIATION.** Extent to which, for at least one week, there is a constant fluctuation of depressed mood and other symptomatology coinciding with the first or second half of day. Generally, if the mood is worse in one part of the day it will be better in the other. However, for occasional subjects who are better in the afternoon and worse both in the morning and the evening, both may be present, or for other subjects there may have been a period when it was clearly worse in the evening. Both items should be rated independently. Rate regardless of regular environmental changes such as feeling better while at work.

Was there any part of the day in which you usually feel better or worse, or didn't it make any difference?

WORSE IN MORNING

- 0 No information
- 1 Not worse in morning or variable
- 2 Minimally or questionably worse
- 3 Mildly worse
- 4 Considerably worse

***Interviewer:** Code as YES if > 2

WORSE IN EVENING

- 0 No information
- 1 Not worse in evening or variable
- 2 Minimally or questionably worse
- 3 Mildly worse
- 4 Considerably worse

***Interviewer:** Code as YES if > 2

21. **EXCESSIVE OR INAPPROPRIATE GUILT** or feelings of self-reproach for things done or not done, including delusions of guilt. (Do not include if simply negative evaluation of self.)

Do you blame yourself for anything you have done or not done?

What about feeling guilty?

Do you feel you have done anything wrong?

(Do you deserve punishment?)

Do you feel you have brought this on yourself?

- 0 No information
- 1 Not at all
- 2 Slight, e.g., occasional feelings of mild self-blame
- 3 Mild, e.g., often feels somewhat guilty about past actions, the significance of which he or she exaggerates, such as consequences of his or her illness
- 4 Moderate, e.g., often feels quite guilty about past actions or feelings of guilt which he or she cannot explain
- 5 Severe, e.g., pervasive feelings of intense guilt, or generalizes feelings of self-blame to most situations
- 6 Extreme, e.g., delusions of guilt, hallucinations in which he or she is accused of having done something terrible, or agonizing constant feelings of guilt

***Interviewer:** Code as YES if >3

MAJOR DEPRESSION IN PARTIAL REMISSION

***Interviewer:** If criteria are already met for MAJOR DEPRESSION, then go to DYSTHYMIA.

22. *Have you recently had a time when you were either much more down or depressed or had even less interest or pleasure in doing things?*

1 = YES 2 = NO

***If YES:** *At that time, did you have many of the problems that I just asked about, like loss of interest or pleasure, social withdrawal, being fidgety or restless or talking or moving more slowly?*

(When was that? How long did it last?)

Comments:

***Interviewer:** Code as YES only if, in the recent past, subject probably had five criterion symptoms and acknowledges some current depressed mood or little interest or pleasure.

DYSTHYMIA

23. *Over the last 2 years, have you often felt down or depressed or had little interest or pleasure in doing things?*

(Have you felt depressed more days than not?)

1 = YES 2 = NO

24. *In the last 2 years, has that often made it hard for you to be with people, take care of things you wanted to do, or get along with people?*

1 = YES 2 = NO

25. *Did a doctor ever say you were manic depressive or give you Lithium?*

1 = YES 2 = NO

***If YES:** *When was that? Do you know why?*

Comments:

PRIOR HISTORY OF DEPRESSION

26. *Before you developed a problem with pain, did you ever have a period of at least 2 weeks where you were bothered by feeling depressed, sad, blue, hopeless... that you didn't care anymore or didn't enjoy anything?*

1 = YES 2 = NO

***If YES:** *At that time, did you also have many other problems, like trouble sleeping, concentrating, feeling tired, poor appetite, little interest in doing things?*

1 = YES 2 = NO

***If YES:** *During that time, did you seek help from anyone, like a doctor, a counsellor, a minister, or even a friend? Did anyone suggest that you seek help? Did you act differently with people, your family, at work, or at school?*

1 = YES 2 = NO

ALCOHOL ABUSE/DEPENDENCE**Section A**

On the questionnaire you said that...

- *If Patient Questionnaire #6 checked YES:** *...you thought you should cut down on your drinking. Why?*
- *If Patient Questionnaire #7 checked YES:** *...someone has complained about your drinking. Who? Why?*
- *If Patient Questionnaire #8 checked YES:** *...you have felt guilty or upset about your drinking. Why?*
- *If Patient Questionnaire #9 checked YES:** *...you had five or more drinks on a single day in the past month. How often have you had that much to drink in the past 6 months? Has that caused any problems?*

Section B

***Assess # 27 to # by any of the following: 1) asking the patient each question; 2) considering the responses given above; or 3) considering other information you know about the patient, such as information obtained from a family member.**

27. *Has a doctor ever suggested that you stop drinking because of a problem with your health?* YES NO

*Count as YES if has continued to drink in the last 6 months after doctor suggested stopping.

Have any of the following happened to you more than one time in the last 6 months?

28. *Were you drinking, high from alcohol, or hung over while you were working, going to school, or taking care of other responsibilities?* YES NO

29. *What about missing or being late for work, school, or other responsibilities because you were drinking or hung over?* YES NO

30. *What about having problems getting along with other people while you were drinking?* YES NO

31. *What about driving a car after having several drinks or after drinking too much?* YES NO

32. *Is at least one of #25 to #29 Yes - OR - do responses in Section A indicate patient has probably had a significant problem with alcohol within the past 6 months?* YES NO

Appendix G
Western Psychiatric Institute and Clinic
Clinical Neuroscience Research Center

NAME _____ ID# _____ DATE _____ TIME _____

STRUCTURED INTERVIEW FOR DSM-IV SLEEP DISORDERS

	QUESTION	NO	Y E S	IF YES, TURN TO PAGE:	Meets DSM-IV Criter
PI	Have you ever had a problem with difficulty falling asleep? Or difficulty staying asleep? Or feeling poorly rested despite an adequate amount of sleep?	<input type="checkbox"/>	<input type="checkbox"/>	1	N Y
PH	Have you ever had a problem with excessive sleepiness including either prolonged nighttime sleep? Or daytime sleep episodes occurring almost daily?	<input type="checkbox"/>	<input type="checkbox"/>	1	N Y
N	Do you have irresistible attacks of sleep?	<input type="checkbox"/>	<input type="checkbox"/>	1	N Y
BRSD	Do you snore or wake up gasping for air?	<input type="checkbox"/>	<input type="checkbox"/>	2	N Y
CRSD	Did the sleep problem occur when your sleep/wake schedule was "out of synch" with other people's or when you had an unusual schedule?	<input type="checkbox"/>	<input type="checkbox"/>	3	N Y
RLS	Have you ever had unpleasant feelings in your legs as bedtime approached?	<input type="checkbox"/>	<input type="checkbox"/>	3	N Y
PLM	Have you ever noticed, or has anyone told you about, jerking leg movements during sleep?	<input type="checkbox"/>	<input type="checkbox"/>	3	N Y
DNOS	Have you ever had any other difficulty with insomnia, sleepiness, or sleep schedules which we have not discussed?	<input type="checkbox"/>	<input type="checkbox"/>	4	N Y
ND	Have you ever awakened from nighttime sleep with terribly frightening dreams?	<input type="checkbox"/>	<input type="checkbox"/>	5	N Y
STD	Have you ever had (or has anyone told you about) abrupt awakenings from sleep beginning with a loud scream?	<input type="checkbox"/>	<input type="checkbox"/>	5	N Y
SWD	Have you had episodes of arising from bed during sleep and walking about?	<input type="checkbox"/>	<input type="checkbox"/>	6	N Y
PNOS	Have you had any other episodes of unusual behavior, events or sensations associated with falling asleep, being asleep or awakening from sleep?	<input type="checkbox"/>	<input type="checkbox"/>	6	N Y

DYSSOMNIAS**I SYMPTOMS****1. PRIMARY INSOMNIA**

Have you ever had a problem with...

...difficulty falling asleep YES NO

OR

...difficulty staying asleep YES NO

OR

...feeling poorly rested despite an adequate amount of sleep? YES NO

How long did this problem last? Duration _____

Is this currently a problem for you? YES NO If NO, Go to **Primary Hypersomnia** ← No Uncertain Yes → If YES, Go to **Impairments Section (p. 7)****2. PRIMARY HYPERSOMNIA**

Have you ever had a problem with excessive sleepiness, including either...

...prolonged (nighttime) sleep YES NO

OR

...daytime sleep episodes occurring almost daily? YES NO

How long did this problem last? Duration _____

Is this currently a problem for you? YES NO If NO, Go to **Narcolepsy** ← No Uncertain Yes → If YES, Go to **Impairments Section (p. 7)****3. NARCOLEPSY**Do you have irresistible attacks of sleep? YES NO Do these occur almost everyday? YES NO Do you feel less sleepy afterwards? YES NO

How long has this been a problem? Duration _____

Is this currently a problem for you? YES NO If NO, Go to **Breathing Related Sleep Disorders** ← No Uncertain Yes → If YES, Continue below

Have you ever had episodes of sudden weakness in your muscles?

Has this weakness generally been on both sides of your body?

(Continued on next page)

Was there any association with strong emotions (such as anger, surprise, laughter)? YES NO

(The weakness may be severe enough to cause a fall or "slump," or may be mild, causing only jaw or hand weakness.)

A. The predominant complaint is difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least one month.

A. The predominant complaint is excessive sleepiness for a least one month (or less if recurrent) as evidenced by either prolonged sleep episodes or daytime sleep episodes that occur almost daily.

A. Irresistible attacks of refreshing sleep that occur daily over a least three months.

B. The presence of one or both of the following:

1. Cataplexy (i.e., brief episodes of sudden bilateral loss of muscle tone, most often in association with intense emotion).

Have you ...		2. Recurrent intrusions of elements of REM sleep into the transition between sleep and wakefulness, as manifested by either hypnogogic/hypnopompic hallucinations or sleep paralysis at the beginning or end of sleep episodes.
...seen images or heard sounds just as you fall asleep or wake up, so that you couldn't quite tell whether it was real or a dream?	YES <input type="checkbox"/> NO <input type="checkbox"/>	
...been unable to move just as you fall asleep or wake up?	YES <input type="checkbox"/> NO <input type="checkbox"/>	

If NO, Go to **Breathing Related Sleep Disorders**

← No Uncertain Yes

→ If YES to A and B, Go to **Impairments Section**

BREATHING-RELATED SLEEP DISORDERS

Have you ever had sleep disruptions which have led to excessive sleepiness or insomnia? (Refer to above sections of interview.)	YES <input type="checkbox"/> NO <input type="checkbox"/>	A. Sleep disruption, leading to excessive sleepiness or insomnia, that is judged to be due to a sleep-related breathing condition (e.g., the sleep apnea or central alveolar hypoventilation syndrome).
After clinical evaluation and polysomnography (if available), determine whether the findings of sleep apnea are present.		

No Uncertain Yes
Continue Below

Do you snore?	YES <input type="checkbox"/> NO <input type="checkbox"/>	*1. Loud snoring (often loud enough to awaken bedpartners or the patient), typically worse when lying on back.
How loudly?		
Is it worse when you sleep in a Particular position?	YES <input type="checkbox"/> NO <input type="checkbox"/>	

Continue Below

Have you, or anyone else ever noticed that you stop breathing during your sleep?	YES <input type="checkbox"/> NO <input type="checkbox"/>	*2. Frequent episodes of breathing cessation, lasting >10 seconds, during sleep.
How long do these episodes last? _____		
How often does this happen in a night? _____		

Continue Below

Have you ever experienced...		*3. Occurrence at some time during the disorder of any of the following:
...headaches upon awakening?	YES <input type="checkbox"/> NO <input type="checkbox"/>	1) Morning headaches 2) Dry mouth on awakening 3) Nocturnal enuresis 4) Fatigue, sleepiness, or confusion on awakening
...dry mouth upon awakening?	YES <input type="checkbox"/> NO <input type="checkbox"/>	
...urinary incontinence during sleep?	YES <input type="checkbox"/> NO <input type="checkbox"/>	
...confusion, disorientation, sleepiness, or tiredness upon awakening?	YES <input type="checkbox"/> NO <input type="checkbox"/>	

If NO, Go to **Circadian Rhythm Sleep Disorders**

← No Uncertain Yes

→ If YES to A, and *1/*2/or *3, Go to **Impairments Section**

***NOTE: Although not diagnostic criteria, items *1-*3 can be used to make a presumptive clinical diagnosis.**

CIRCADIAN RHYTHM SLEEP DISORDERS

Has your sleep-wake schedule been "out of synch" with other people's, or have you had an unusual sleep-wake schedule which resulted in sleepiness or insomnia?	A. A persistent or recurrent pattern of sleep disruption leading to excessive sleepiness or insomnia that is due to a mismatch between the
--	--

YES NO sleep-wake schedule required by a person's environment and his or her circadian sleep-wake pattern.

If NO, Go to **Restless Legs Syndrome** ← No Uncertain Yes → If YES, Continue below

Specify type:

- | | |
|--|---|
| <p>1. Was the sleep schedule problem characterized by falling asleep and waking up much later than you desired? YES <input type="checkbox"/> NO <input type="checkbox"/></p> <p>2. Was the sleep schedule problem associated with travel across time zones? YES <input type="checkbox"/> NO <input type="checkbox"/></p> <p>3. Did the sleep schedule problem occur while you were working night shift, rotating shifts, or other unusual hours? YES <input type="checkbox"/> NO <input type="checkbox"/></p> <p>4. Was the sleep schedule problem different than what we have just talked about? YES <input type="checkbox"/> NO <input type="checkbox"/></p> | <p>1. Delayed Sleep Phase Type: a persistent pattern of late sleep onset and late awakening times, with an inability to fall asleep and awaken at a desired earlier time.</p> <p>2. Jet Lag Type: sleepiness and alertness that occur at an inappropriate time of day relative to local time, occurring after repeated travel across more than one time zone.</p> <p>3. Shift Work Type: insomnia during the major sleep period or excessive sleepiness during the major awake period associated with night shift work or frequently changing shift work.</p> <p>4. Unspecified Type: (e.g., advanced sleep phase, non-24-hour sleep-wake pattern, irregular sleep-wake pattern, or other unspecified pattern).</p> |
|--|---|

Go to **Impairments Section** (p. 7)

RESTLESS LEGS SYNDROME NOTE: This is not a specific diagnosis in DSM-IV, but is a common form of dyssomnia which may have specific diagnostic and treatment implications

- | | |
|---|--|
| <p>Have you ever had unpleasant feelings in your legs as bedtime approached? YES <input type="checkbox"/> NO <input type="checkbox"/></p> <p>Did these feelings include crawling or electric sensations? YES <input type="checkbox"/> NO <input type="checkbox"/></p> <p>Are the sensations relieved by moving your legs or walking? YES <input type="checkbox"/> NO <input type="checkbox"/></p> <p>How often does this occur? _____</p> <p>How long did the problem last? Duration _____</p> <p>Is this currently a problem for you? YES <input type="checkbox"/> NO <input type="checkbox"/></p> | <p>A. Uncomfortable sensations in the lower limbs which increase prior to bedtime, and are relieved by movement. The problem occurs nearly every day for at least one month.</p> |
|---|--|

If NO, Go to **PLM Sleep Disorder** ← No Uncertain Yes → If YES, Continue below

- | | |
|--|---|
| <p>Do these feelings ever cause you to have difficulty falling or staying asleep? YES <input type="checkbox"/> NO <input type="checkbox"/></p> | <p>B. The uncomfortable sensations cause difficulty falling asleep or awakenings after sleep onset.</p> |
|--|---|

If NO, Go to **PLM Sleep Disorder** ← No Uncertain Yes → If YES, Go to **Impairments Section** (p. 7)

PERIODIC LIMB MOVEMENT SLEEP DISORDER NOTE: This is not a specific diagnosis in DSM-IV, but is a common form of dyssomnia which may have specific diagnostic and treatment implications.

- | | |
|---|--|
| <p>Based on responses, does the patient have a current or past complaint of insomnia or hypersomnia? YES <input type="checkbox"/> NO <input type="checkbox"/></p> | <p>A. A prominent complaint of insomnia or hypersomnia nearly every day for a least one month.</p> |
|---|--|

If NO, Go to **Dyssomnia NOS** ← No Uncertain Yes → If YES, Continue Below

- | | |
|--|---|
| <p>Have you ever noticed, or has anyone told you about, jerking leg movements during your sleep? YES <input type="checkbox"/> NO <input type="checkbox"/></p> <p>What sort of movements were these? _____</p> <p>Were these movements repetitive? YES <input type="checkbox"/> NO <input type="checkbox"/></p> | <p>B. Repetitive, rhythmic, small-amplitude leg movements characterized by extension of the toe, and partial flexion of the ankle, knee, and sometimes hip.</p> |
|--|---|

Continue below

After polysomnography, determine whether the patient has *C. Polysomnographic monitoring demonstrates:

PARASOMNIAS**I SYMPTOMS****NIGHTMARE DISORDER**

1. Have you ever been awakened from sleep or naps with frightening dreams? YES NO

How often do these dreams occur? _____

How well do you remember the dreams when you awaken?

What are the dreams about? _____

During what part of your sleep period (night) do these tend to occur? _____

A. Repeated awakenings from the major sleep period or naps with detailed recall of extended and extremely frightening dreams, usually involving threats to survival, security, or self-esteem. The awakenings generally occur during the second half of the sleep period.

If NO, Go to **Sleep Terror Disorder**

← No Uncertain Yes → If YES, Continue below

2. When you have these dreams, do you awaken and become alert quickly? YES NO

Have others ever told you you appear confused or disoriented during these events? YES NO

B. On awakening from the frightening dreams, the person rapidly becomes oriented and alert (in contrast to the confusion and disorientation seen in Sleep Terror Disorder and some forms of epilepsy).

If NO, Go to **Sleep Terror Disorder**

← No Uncertain Yes → If YES, Go to **Impairments Section (p. 7)**

SLEEP TERROR DISORDER

1. Have you ever had (or has anyone ever told you that you had) abrupt awakenings from sleep, beginning with a loud scream? YES NO

How often? _____

When during your sleep has this usually occurred?

A. Recurrent episodes of abrupt awakening from sleep, usually occurring during the first third of the major sleep episode and beginning with a panicky scream.

If NO, Go to **Sleepwalking Disorder**

← No Uncertain Yes → If YES, Continue below

2. Have you felt anxious or appeared anxious to others during these episodes? YES NO

Have you been, or have others told you that you were, physically or emotionally aroused during these episodes? For instance, did you have palpitations, rapid heart beat, rapid breathing, or sweating? YES NO

B. Intense fear and signs of autonomic arousal, such as tachycardia, rapid breathing, and sweating, during each episode.

If NO, Go to **Sleepwalking Disorder**

← No Uncertain Yes → If YES, Continue below

3. Has anyone told you that you could not be fully awakened or comforted during these episodes? YES NO

C. Relative unresponsiveness to efforts of others to comfort the person during the episode.

If NO, Go to **Sleepwalking Disorder**

← No Uncertain Yes → If YES, Continue below

4. Do you recall any dreams with these episodes? YES NO

Do you recall the events themselves? YES NO

D. No detailed dream is recalled, and there is amnesia for the episode.

If NO, Go to **Impairments Section (p. 7)**

← No Uncertain Yes → If YES, Go to **Sleepwalking Disorder**

SLEEPWALKING DISORDER

1. Have you had episodes of arising from bed during

A. Repeated episodes of rising from bed during

sleep and walking about?	YES <input type="checkbox"/>	NO <input type="checkbox"/>	sleep and walking about, usually occurring during the first third of the major sleep episode.
When during sleep did this occur?	_____		
How often does this occur?	_____		

If NO, Go to **Parasomnia NOS** ← No Uncertain Yes → If YES, Continue below

2. During these episodes, have others described you as...			B. While sleepwalking, the person has a blank, staring face, is relatively unresponsive to the efforts of others to communicate with him/her, and can be awakened only with great difficulty.
...having a blank, staring face?	YES <input type="checkbox"/>	NO <input type="checkbox"/>	
...being difficult to communicate with?	YES <input type="checkbox"/>	NO <input type="checkbox"/>	
...being awakened only with great difficulty?	YES <input type="checkbox"/>	NO <input type="checkbox"/>	

If NO, Go to **Parasomnia NOS** ← No Uncertain Yes → If YES, Continue below

3. When you finally awaken (according to what others have told you), can you recall these episodes?	YES <input type="checkbox"/>	NO <input type="checkbox"/>	C. On awakening (either from the sleep walking episode or the next morning), the person has amnesia for the episode.
---	------------------------------	-----------------------------	--

If NO, Continue below ← No Uncertain Yes → If YES, Go to **Parasomnia NOS**

4. If you awaken, does it take you a while to become fully awake and alert?	YES <input type="checkbox"/>	NO <input type="checkbox"/>	D. Within several minutes after awakening from the sleep walking episode, there is no impairment of mental activity or behavior (although there may initially be a short period of confusion or disorientation).
When you awaken, are you alert and oriented within several minutes?	YES <input type="checkbox"/>	NO <input type="checkbox"/>	

If NO, Go to **Parasomnia NOS** ← No Uncertain Yes → If YES, Go to **Impairments Section (p. 7)**

PARASOMNIA NOT OTHERWISE SPECIFIED

1. Have you had any other episodes of unusual behavior, physical events, or peculiar sensations associated with falling asleep, being asleep, or awakening from sleep?	YES <input type="checkbox"/>	NO <input type="checkbox"/>	A. This category is for disturbances that are characterized by abnormal behavioral or physiological events during sleep or sleep-wake transitions, but that do not meet criteria for a more specific Parasomnia.
--	------------------------------	-----------------------------	--

If NO, INTERVIEW COMPLETED ← No Uncertain Yes → If YES, Go to **Impairments Section (p. 7)**

**COMPLETE SECTIONS II - IV FOR EACH INDIVIDUAL SLEEP DISORDER
IDENTIFIED IN THE "SYMPTOMS" SECTION ABOVE**

II IMPAIRMENTS

Sleep Disorder #1: _____

How much did the sleep problem bother you?

The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

During (*Sleep Disorder*) did you have...

...difficulty getting along with people YES NO

OR

...difficulty functioning at work or home (e.g., sleepiness, irritability, poor concentration, poor memory) YES NO

OR

...limitation or disruption of daytime work and activities? YES NO

(Consider injuries or problems with bed partner as part of difficulties)

If NO, Go to next disorder symptom Section

← No Uncertain Yes → If YES, Go to **Medical/Psychiatric Criteria** (p. 8)

Sleep Disorder #2: _____

How much did the sleep problem bother you?

The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

During (*Sleep Disorder*) did you have...

...difficulty getting along with people YES NO

OR

...difficulty functioning at work or home (e.g., sleepiness, irritability, poor concentration, poor memory) YES NO

OR

...limitation or disruption of daytime work and activities? YES NO

(Consider injuries or problems with bed partner as part of difficulties)

If NO, Go to next disorder symptom Section

← No Uncertain Yes → If YES, Go to **Medical/Psychiatric Criteria** (p. 8)

Sleep Disorder #3: _____

How much did the sleep problem bother you?

The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

During (*Sleep Disorder*) did you have...

...difficulty getting along with people YES NO

OR

...difficulty functioning at work or home (e.g., sleepiness, irritability, poor concentration, poor memory) YES NO

OR

...limitation or disruption of daytime work and activities? YES NO

(Consider injuries or problems with bed partner as part of difficulties)

If NO, Go to next disorder symptom Section

← No Uncertain Yes → If YES, Go to **Medical/Psychiatric Criteria** (p. 8)

III MEDICAL/PSYCHIATRIC CRITERIA

SUBSTANCE-INDUCED/DRUG-RELATED

Within one month of (*the sleep disturbance*), were you using or withdrawing from ...

...prescribed medications? YES NO
 ...nonprescribed medications? YES NO
 ...alcohol? YES NO
 ...other drugs? YES NO

The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication).

Specify substance(s), if any:

To determine whether the insomnia was due to the substance, refer to all available psychiatric and medical history.

If NO, Continue below

← No Uncertain Yes

→ *If YES, consider Substance-Induced Sleep Disorder and Go to **Episode History** (p. 9)*

SECONDARY TO A MEDICAL CONDITION

At the time of (*the sleep disturbance*), were you suffering from any illness, chronic medical condition, or physical disability (including pain, immobility, forced bedrest)?

YES NO

...or a Secondary Sleep Disorder.
Describe:

To determine whether the sleep disturbance was due to the medical disorder, refer to all available medical history, laboratory results, and physical exam findings.

If NO, Continue below

← No Uncertain Yes

→ *If YES, consider Sleep Disorder Due to a General Medical Condition and Go to **Episode History** (p. 9)*

SECONDARY TO A MENTAL DISORDER

At the time of (*the sleep disturbance*), were you being treated for or suffering from depression, anxiety, or any other psychological symptoms? (Questions regarding psychiatric disorders)

YES NO

...or mental disorder.
Describe:

To determine whether the sleep disturbance was due to, or better accounted for by a mental disorder, refer to all available psychiatric history sources.

*If NO, consider Primary Sleep Disorder and Go to **Episode History** (p. 9)*

← No Uncertain Yes →

*If YES, consider Sleep Disorder Related to Another Mental Disorder and Go to **Episode History** (p. 9)*

IV EPISODE HISTORY

Complete specific criteria questions for the most likely diagnosis: specify-

At what age did (*the sleep problem*) first occur?

How long did this first episode last (in weeks)?

Was this initial episode diagnosed properly?

How many separate time have you experienced this type of sleep disturbance?

What was the date of onset of the most recent episode?

[If no current episode] What was the date of offset of your most recent episode?

Age at onset of initial episode: _____ years

Length of initial episode: _____ weeks

Diagnosed? N Y

Number of episodes: _____

Onset Date of most recent
(includes current)

Offset Date of most recent
(Not Current)

Complete specific criteria questions for the next likely diagnosis: specify-

At what age did (*the sleep problem*) first occur?

How long did this first episode last (in weeks)?

Was this initial episode diagnosed properly?

How many separate time have you experienced this type of sleep disturbance?

What was the date of onset of the most recent episode?

[If no current episode] What was the date of offset of your most recent episode?

Age at onset of initial episode: _____ years

Length of initial episode: _____ weeks

Diagnosed? N Y

Number of episodes: _____

Onset Date of most recent
(includes current)

Offset Date of most recent
(Not Current)

Complete specific criteria questions for the next likely diagnosis: specify-

At what age did (*the sleep problem*) first occur?

How long did this first episode last (in weeks)?

Was this initial episode diagnosed properly?

How many separate time have you experienced this type of sleep disturbance?

What was the date of onset of the most recent episode?

[If no current episode] What was the date of offset of your most recent episode?

Age at onset of initial episode: _____ years

Length of initial episode: _____ weeks

Diagnosed? N Y

Number of episodes: _____

Onset Date of most recent
(includes current)

Offset Date of most recent
(Not Current)

Interviewer: Global rating for severity of insomnia:

Insomnia. Sleep disturbance, including initial, middle, and terminal difficulty in getting to sleep or staying asleep. Do not rate if feels no need for sleep. Take into account the estimated number of hours slept and subjective sense of loss of sleep. If the subject is using medication, ask what he/she thinks it would be like without medication.

*Have you had trouble sleeping?
(What about falling asleep?)
(...or waking up in the middle of the night?)
(...or early in the morning before you want to get up?)*

(How bad does it get?)

- 0 No information
- 1 Not at all, or feels no need for any sleep
- 2 Slight, e.g., occasional difficulty
- 3 Mild, e.g., often has some significant difficulty
- 4 Moderate, e.g., usually has considerable difficulty
- 5 Severe, e.g., almost always has great difficulty
- 6 Extreme, e.g., claims he/she almost never sleeps and feels exhausted the next day

If ≥ 3 :

Note types(s):

___ Initial insomnia: Difficulty falling asleep

___ Middle insomnia: Difficulty staying asleep or returning to sleep, preceded and followed by sleep

___ Terminal insomnia: Difficulty staying asleep the usual amount of time (beyond 5 hours), or final awakening after less than 5 hours of sleep

___ Melancholia criteria: Often awakens at least 2 hours before usual time of awakening

(What about during the past week?)

PAST WEEK: 0 1 2 3 4 5 6

Appendix H

Demographic Information

To help us understand your current situation, please complete the following as fully as possible.

BACKGROUND INFORMATION

1. Date: _____

Day/Month/Year

2. Name: _____

First

Middle

Last

(Maiden Name)

3. Sex: Male Female

4. Age: _____ Date of Birth: _____
In Years *Day/Month/Year*

5. We try to be sensitive to issues of ethnic diversity. In broad terms, how would you categorize your ethnic background:

Caucasian origin

First Nations origin

Asian origin

Other (*please specify*):

African origin

6. Preferred language for service:

English French Either

7. What is your **current** employment status?

Working - Full-time

Unemployed - Because of pain

Working - Part-time

Unemployed - Not because of pain

Sick leave

Retired

Student/retraining - Full-time

Disability leave - Temporary

Student/retraining - Part-time

Disability leave - Permanent

Homemaker/parenting

Other (*please specify*):

8. Are you involved in any legal action or appeal in regard to an accident, or in regard to your compensation or pension status?

No Yes

If **YES**, please describe:

9. What is your educational background? Check () the highest level achieved.

<input type="checkbox"/> Some primary school	<input type="checkbox"/> Some college/university
<input type="checkbox"/> Completed primary school	<input type="checkbox"/> College/university graduate
<input type="checkbox"/> Some high school	<input type="checkbox"/> Trade certification
<input type="checkbox"/> Completed high school	<input type="checkbox"/> Postgraduate university

THANK YOU

Appendix I

Medication Quantification Scale (MQS)

Please indicate the type and amount of medications (prescription and over-the-counter) you have taken today (in total):

Medication (please be as specific as possible):	Dose per pill:	Number of pills taken:
1. _____	_____	_____
2. _____	_____	_____
3. _____	_____	_____
4. _____	_____	_____
5. _____	_____	_____
6. _____	_____	_____
7. _____	_____	_____
8. _____	_____	_____
9. _____	_____	_____

Appendix J

PAIN AND MEDICAL HISTORY

1. a) Where do you experience the most pain? Please check (✓) only one.

- | | |
|--|--|
| <input type="checkbox"/>]Head, face, mouth | <input type="checkbox"/>]Lower back, lumbar spine |
| <input type="checkbox"/>]Neck (cervical) region | <input type="checkbox"/>]Legs, feet |
| <input type="checkbox"/>]Shoulders | <input type="checkbox"/>]Pelvic region |
| <input type="checkbox"/>]Arms, hands | <input type="checkbox"/>]Genital region |
| <input type="checkbox"/>]Chest | <input type="checkbox"/>]All over body |
| <input type="checkbox"/>]Abdominal Region | <input type="checkbox"/>]Joints |
| <input type="checkbox"/>]Upper back | <input type="checkbox"/>]Other (<i>please specify</i>): |
-

b) If you have pain in other areas as well, please check (✓) all that apply.

- | | |
|--|--|
| <input type="checkbox"/>]Head, face, mouth | <input type="checkbox"/>]Lower back, lumbar spine |
| <input type="checkbox"/>]Neck (cervical) region | <input type="checkbox"/>]Legs, feet |
| <input type="checkbox"/>]Shoulders | <input type="checkbox"/>]Pelvic region |
| <input type="checkbox"/>]Arms, hands | <input type="checkbox"/>]Genital region |
| <input type="checkbox"/>]Chest | <input type="checkbox"/>]All over body |
| <input type="checkbox"/>]Abdominal Region | <input type="checkbox"/>]Joints |
| <input type="checkbox"/>]Upper back | <input type="checkbox"/>]Other (<i>please specify</i>): |
-

2.a) When did your current pain problem start? _____ (*Month/Year*)

b) How long have you had your current pain problem? _____ (*Months or Years*)

c) How did your current pain problem begin?

- | | |
|--|--|
| <input type="checkbox"/>]Motor vehicle accident | <input type="checkbox"/>]Pain just began |
| <input type="checkbox"/>]Accident at home | <input type="checkbox"/>]After an illness |
| <input type="checkbox"/>]Accident at work | <input type="checkbox"/>]Other (<i>please specify</i>): _____ |
| <input type="checkbox"/>]Other accident | |

Please describe in detail how your pain problem began (continue on back of page if necessary):

Appendix L

Pain Severity Ratings

Name: _____ Date: _____

Rate the severity of your pain at each of the times indicated below. Circle the number that best corresponds to your pain level at that time.

Pain Rating

	No Pain											Pain as Bad as could be
1. First thing in the morning.	0	1	2	3	4	5	6	7	8	9	10	
2. At noon.	0	1	2	3	4	5	6	7	8	9	10	
3. At suppertime.	0	1	2	3	4	5	6	7	8	9	10	
4. At bedtime.	0	1	2	3	4	5	6	7	8	9	10	

Appendix M

Pittsburgh Sleep Quality Index (PSQI)

The following questions relate to your usual sleep habits during the past month *only*. Your answers should indicate the most accurate reply for the *majority* of days and nights in the past month. Please answer all questions.

1. During the past month, when have you usually gone to bed at night?

USUAL BED TIME _____

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

NUMBER OF MINUTES _____

3. During the past month, when have you usually got up in the morning?

USUAL GETTING UP TIME _____

4. During the past month, how many hours of actual sleep did you get at night?
(This may be different than the number of hours you spend in bed)

HOURS OF SLEEP PER NIGHT _____

For each of the remaining questions, check the best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you...

- (a) Cannot get to sleep within 30 minutes

_____ Not during the past month
 _____ Less than once a week
 _____ Once or twice a week
 _____ Three or more times a week

- (b) Wake up in the middle of the night or early morning

_____ Not during the past month
 _____ Less than once a week
 _____ Once or twice a week
 _____ Three or more times a week

- (c) Have to get up to use the bathroom

_____ Not during the past month
 _____ Less than once a week
 _____ Once or twice a week
 _____ Three or more times a week

(d) Cannot breathe comfortably

- Not during the past month
 Less than once a week
 Once or twice a week
 Three or more times a week

(e) Cough or snore loudly

- Not during the past month
 Less than once a week
 Once or twice a week
 Three or more times a week

(f) Feel too cold

- Not during the past month
 Less than once a week
 Once or twice a week
 Three or more times a week

(g) Feel too hot

- Not during the past month
 Less than once a week
 Once or twice a week
 Three or more times a week

(h) Had bad dreams

- Not during the past month
 Less than once a week
 Once or twice a week
 Three or more times a week

(i) Have pain

- Not during the past month
 Less than once a week
 Once or twice a week
 Three or more times a week

(j) Other reason(s), please describe: _____

How often during the past month have you had trouble sleeping because of this?

- Not during the past month
 Less than once a week
 Once or twice a week
 Three or more times a week

6. During the past month, how would you rate your sleep quality overall?
- Very good
 - Fairly good
 - Fairly bad
 - Very bad
7. During the past month, how often have you taken medicine (prescribed or 'over-the-counter') to help you sleep?
- Not during the past month
 - Less than once a week
 - Once or twice a week
 - Three or more times a week
8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?
- Not during the past month
 - Less than once a week
 - Once or twice a week
 - Three or more times a week
9. During the past month, how much of a problem has it been for you to keep enough enthusiasm to get things done?
- No problem at all
 - Only a very slight problem
 - Somewhat of a problem
 - A very big problem
10. Do you have a bed partner or roommate?
- No bed partner or roommate
 - Partner/roommate in other room
 - Partner in same room, but not same bed
 - Partner in same bed

Appendix N

Sleep Hygiene Usual Practice

For each of the following behaviours state the number of days per week (0-7) that you engage in that activity or have that experience. Base your answers on what you would consider an average week for yourself.

Indicate the number of days or nights in an average week you:

1. Take a nap _____
2. Go to bed hungry _____
3. Go to bed thirsty _____
4. Smoke more than one pack of cigarettes _____
5. Use sleeping medications (prescription or over-the-counter) _____
6. Drink beverages containing caffeine (e.g., coffee, tea, colas) within 4 hours of bedtime _____
7. Drink more than 3 ounces of alcohol (e.g., 3 mixed drinks, 3 beers, or 3 glasses of wine) within 2 hours of bedtime _____
8. Take medications/drugs with caffeine within 4 hours of bedtime _____
9. Worry as you prepare your bed about your ability to sleep _____
10. Worry during the day about your ability to sleep at night _____
11. Use alcohol to facilitate sleep _____
12. Exercise strenuously within 2 hours of bedtime _____
13. Have your sleep disturbed by light _____
14. Have your sleep disturbed by noise _____
15. Have your sleep disturbed by your bed partner _____ (put NA if no partner)
16. Sleep approximately the same length of time each night _____
17. Set aside time to relax before bedtime _____

18. Exercise in the afternoon or early evening _____

19. Have a comfortable nighttime temperature in your bed/bedroom _____

Appendix O
Daily Sleep Diary

Today's date is: _____ (day, month, year)

To be completed in the morning

1. What time did you go to bed? (circle am or pm) _____ am/pm
2. What time did you get out of bed? _____ am/pm
3. Approximately how many hours of sleep did you get last night? (to the nearest quarter hour) _____ hours
4. How long did it take you to fall asleep last night after you turned out the light? _____ minutes
5. How many times did you wake up during the night? _____ times
6. In the morning, did you awaken at the time you wanted to?
 - _____ Earlier
 - _____ On time
 - _____ Later
7. At what time did you wake up for the last time? (circle am or pm) _____ am/pm
8. Please rate the overall **quality** of your sleep last night (circle one):

0	1	2	3	4	5	6	7	8	9	10
As poor as										As good as
can be										can be
9. Please rate how **rested** you felt this morning upon awakening:

0	1	2	3	4	5	6	7	8	9	10
As poorly rested										As well-rested
as can be										as can be

Appendix P

Sleep Hygiene Daily Diary

For each of the following behaviours state whether or not you engaged in that activity or had that experience yesterday or last night. Mark 'Y' for yes, and 'N' for no.

Yesterday or last night, did you:

1. Take a nap _____
2. Go to bed hungry _____
3. Go to bed thirsty _____
4. Smoke more than one pack of cigarettes _____
5. Use sleeping medications (prescription or over-the-counter) _____
6. Drink beverages containing caffeine (e.g., coffee, tea, colas) within 4 hours of bedtime _____
7. Drink more than 3 ounces of alcohol (e.g., 3 mixed drinks, 3 beers, or 3 glasses of wine) within 2 hours of bedtime _____
8. Take medications/drugs with caffeine within 4 hours of bedtime _____
9. Worry as you prepared for bed about your ability to sleep _____
10. Worry during the day about your ability to sleep at night _____
11. Use alcohol to facilitate sleep _____
12. Exercise strenuously within 2 hours of bedtime _____
13. Have your sleep disturbed by light _____
14. Have your sleep disturbed by noise _____
15. Have your sleep disturbed by your bed partner _____ (put NA if no partner)
16. Sleep approximately the same length of time as you do most nights _____
17. Set aside time to relax before bedtime _____
18. Exercise in the afternoon or early evening _____
19. Have a comfortable nighttime temperature in your bed/bedroom _____

If you did you take any naps yesterday, please indicate when you napped, and for how long:

From _____ to _____ (_____ minutes)

From _____ to _____ (_____ minutes)

From _____ to _____ (_____ minutes)

11. In order to be alert and function well during the day, I am better off taking a sleeping pill rather than having a poor night's sleep.

1	2	3	4	5	6	7	8	9	10
Strongly Disagree								Strongly Agree	

12. When I feel irritable, depressed, or anxious during the day, it is mostly because I did not fall asleep the night before.

1	2	3	4	5	6	7	8	9	10
Strongly Disagree								Strongly Agree	

13. Because my bed partner falls asleep as soon as his or her head hits the pillow and stays asleep through the night, I should be able to do so too.

1	2	3	4	5	6	7	8	9	10
Strongly Disagree								Strongly Agree	

14. I feel that insomnia is basically the result of aging, and there isn't much that can be done about this problem.

1	2	3	4	5	6	7	8	9	10
Strongly Disagree								Strongly Agree	

15. I am sometimes afraid of dying in my sleep.

1	2	3	4	5	6	7	8	9	10
Strongly Disagree								Strongly Agree	

16. When I have a good night's sleep, I know that I will have to pay for it on the following night.

1	2	3	4	5	6	7	8	9	10
Strongly Disagree								Strongly Agree	

Appendix R

Sleep Hygiene Knowledge Scale

This is a survey of the effect of selected daytime behaviours upon sleep. We are interested in knowing your opinion about whether any of these daytime behaviours influence the quality and/or quantity of sleep. For the following list of behaviours, please indicate your opinion as to the extent of the general effect, if any, that each behaviour may have on nightly sleep. Please use the following scale and answer each item by writing the appropriate number in the space provided. Note that numbers 5, 6, and 7 indicate degrees of *disruption* of sleep.

Beneficial to sleep			No effect	Disruptive to sleep		
1	2	3	4	5	6	7
very	moderately	mildly		mildly	moderately	very

What effect do these behaviours have on sleep?

1. Daytime napping _____
2. Going to bed hungry _____
3. Going to bed thirsty _____
4. Smoking more than one pack of cigarettes a day _____
5. Using sleep medication regularly _____
6. Exercising strenuously within 2 hours of bedtime _____
7. Sleeping approximately the same length of time each night _____
8. Setting aside time to relax before bedtime _____
9. Consuming food, beverages, or medications containing caffeine _____
10. Exercising in the afternoon or the early evening _____
11. Waking up at the same time each day _____
12. Going to bed at the same time each day _____
13. Drinking 3 ounces of alcohol in the evening (e.g., 3 mixed drinks, 3 beers, 3 glasses of wine) _____

Appendix S

Caffeine Knowledge

For each item on the following list, indicate whether you believe it contains caffeine or another stimulant by placing a Y (yes) or an N (no) in the space provided. If you are not sure, make your best guess. If you have never heard of an item please place an X in the space.

_____ 7-Up soft drink	_____ lemonade	_____ Mountain Dew
_____ regular tea	_____ root beer	_____ cola soft drinks
_____ Dristan cold remedy	_____ chocolate cake	_____ Dexatrim diet pills
_____ aspirin	_____ regular coffee	_____ Tylenol
_____ Dr. Pepper soft drink	_____ Excedrin	_____ Aqua Ban diuretic
_____ Midol menstrual relief	_____ Sudafed decongestant	_____ Sprite soft drink

Appendix T

PSAS

During the pre-sleep period last night (in bed with the lights out before falling asleep for the first time), did you have any of the following experiences? Please indicate (by circling the appropriate number) the degree to which you experienced each of those listed below. Do not include what you experienced during the middle of the night if you awakened after falling asleep.

	Not at all	A little	Moderately	A lot	Extremely
1. Heart racing, pounding, or beating irregularly	1	2	3	4	5
2. A jittery, nervous feeling in your body	1	2	3	4	5
3. Worry about falling asleep	1	2	3	4	5
4. Review or ponder events of the day	1	2	3	4	5
5. Shortness of breath or labored breathing	1	2	3	4	5
6. Depressing or anxious thoughts	1	2	3	4	5
7. A tight, tense feeling in your muscles	1	2	3	4	5
8. Worry about problems other than sleep	1	2	3	4	5
9. Being mentally alert, active	1	2	3	4	5
10. Cold feeling in your hands, feet, or your body in general	1	2	3	4	5
11. Can't shut off your thoughts	1	2	3	4	5
12. Have stomach upset (knot or nervous feeling in stomach, heartburn, nausea, gas, etc.)	1	2	3	4	5

	Not at all	A little	Moderately	A lot	Extremely
13. Perspiration in palms of your hands or other parts of your body	1	2	3	4	5
14. Thoughts keep running through your head	1	2	3	4	5
15. Dry feeling in mouth or throat	1	2	3	4	5
16. Distracted by sounds, noise in the environment (e.g., ticking clock, house noises, traffic)	1	2	3	4	5

Appendix U

PANAS

How Do You Feel Right Now?

How do you feel right now? For each item below, please circle the number that best corresponds to how you are feeling right now.

Do you feel:	Slightly or Not at all	A Little	Moder- ately	Quite a Bit	Very Much
1. Active	1	2	3	4	5
2. Afraid	1	2	3	4	5
3. Alert	1	2	3	4	5
4. Ashamed	1	2	3	4	5
5. Attentive	1	2	3	4	5
6. Determined	1	2	3	4	5
7. Distressed	1	2	3	4	5
8. Enthusiastic	1	2	3	4	5
9. Excited	1	2	3	4	5
10. Guilty	1	2	3	4	5
11. Hostile	1	2	3	4	5
12. Inspired	1	2	3	4	5
13. Interested	1	2	3	4	5
14. Irritable	1	2	3	4	5
15. Jittery	1	2	3	4	5
16. Nervous	1	2	3	4	5
17. Proud	1	2	3	4	5
18. Scared	1	2	3	4	5
19. Strong	1	2	3	4	5
20. Upset	1	2	3	4	5

Total PA:

Total NA: