Combining cognitive behavioral therapy for insomnia and chronic spinal pain within physical therapy: a practical guide for the implementation of an integrated approach

<table>
<thead>
<tr>
<th>Journal:</th>
<th>Physical Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manuscript ID</td>
<td>PTJ-2021-0327.R2</td>
</tr>
<tr>
<td>Manuscript Category:</td>
<td>Perspective</td>
</tr>
<tr>
<td>Section:</td>
<td>Pain Management</td>
</tr>
<tr>
<td>Keywords:</td>
<td>Insomnia, Cognitive Behavioral Therapy, Chronic Spinal Pain</td>
</tr>
</tbody>
</table>
Combining cognitive behavioral therapy for insomnia and chronic spinal pain within physical therapy: a practical guide for the implementation of an integrated approach

E. Van Looveren, PT, MT, MSc, Pain in Motion international Research Group; Department of Physiotherapy, Human Physiology and Anatomy (KIMA), Faculty of Physical Education, and Physiotherapy, Vrije Universiteit Brussel, Brussels, Belgium; and Department of Rehabilitation Sciences, Faculty of Medicine and Health Sciences, Ghent University, Campus UZ Ghent 3 B3, Corneel Heymanslaan 10, BE-9000 Ghent, Belgium. Address all correspondence to Dra. Van Looveren at: eveline.vanlooveren@ugent.be.

M. Meeus, PT, PhD, Pain in Motion international Research Group, Department of Rehabilitation Sciences, Faculty of Medicine and Health Sciences, Ghent University, Ghent, Belgium, and Department of Rehabilitation Sciences and Physiotherapy (REVAKI), Faculty of Medicine and Health Sciences, University of Antwerp, Antwerp, Belgium.

B. Cagnie, PT, MT, PhD, Department of Rehabilitation Sciences, Faculty of Medicine and Health Sciences, Ghent University, Ghent, Belgium.

K. Ickmans, PT, PhD, Pain in Motion International Research Group (PAIN), Department of Physiotherapy, Human Physiology and Anatomy (KIMA), Faculty of Physical Education and Physiotherapy, Vrije Universiteit Brussel, Brussels, Belgium; Department of Physical Medicine and Physiotherapy, University Hospital Brussels, Brussels, Belgium; Research Foundation – Flanders (FWO), Brussels, Belgium.

T. Bilterys, PT, MSc, Pain in Motion international Research Group, Department of Physiotherapy, Human Physiology and Anatomy (KIMA), Faculty of Physical Education, and
Physiotherapy, Vrije Universiteit Brussel, Brussels, Belgium and Department of Rehabilitation Sciences, Faculty of Medicine and Health Sciences, Ghent University, Ghent, Belgium.

A. Malfliet, PT, PhD, Pain in Motion international Research Group, Department of Physiotherapy, Human Physiology and Anatomy (KIMA), Faculty of Physical Education and Physiotherapy, Vrije Universiteit Brussel, Brussels, Belgium, and Department of Physical Medicine and Physiotherapy, University Hospital Brussels, Brussels, Belgium.

D. Goubert, PT, PhD, Pain in Motion international Research Group, Department of Rehabilitation Sciences, Faculty of Medicine and Health Sciences, Ghent University, Ghent, Belgium.

J. Nijs, PT, MT, PhD, Pain in Motion international Research Group (PAIN), Department of Physiotherapy, Human Physiology and Anatomy (KIMA), Faculty of Physical Education and Physiotherapy, Vrije Universiteit Brussel, Brussels, Belgium, Department of Physical Medicine and Physiotherapy, University Hospital Brussels, Brussels, Belgium, and Institute of Neuroscience and Physiology, University of Gothenburg, Gothenburg, Sweden.

L. Danneels, PT, PhD, Department of Rehabilitation Sciences, Faculty of Medicine and Health Sciences, Ghent University, Ghent, Belgium.

M. Moens, MD, PhD, Department of Neurosurgery and Radiology, University Hospital Brussels, Brussels, Belgium, and Center of Neurosciences (C4N), Vrije Universiteit Brussel, Brussels, Belgium.

O. Mairesse, PhD, Department of Psychology - Brain, Body and Cognition (BBCO), Vrije Universiteit Brussel, Brussels, Belgium, and Brugmann University Hospital, Sleep laboratory and Unit for Clinical Chronobiology U78, Brussels, Belgium.
ABSTRACT

The majority of people suffering from nonspecific chronic spinal pain (nCSP) report comorbid insomnia. However, in current treatment strategies for nCSP, insomnia is usually not addressed. Considering the bidirectional interaction between pain and sleep and its underlying psychophysiological mechanisms, insomnia may increase the risk of developing adverse physical and psychological health outcomes and should thus no longer be left untreated. As suggested by previous pilot studies, adding cognitive behavioral therapy for insomnia (CBT-I) to the contemporary evidence-based biopsychosocial physical therapy approach, may also improve pain outcomes in nCSP. This manuscript aims at providing practical guidelines on how hybrid physical therapy, including the combination of following components: (1) pain neuroscience education (i.e., to reconceptualize pain) and cognition-targeted exercise therapy (i.e., graded exposure to functional daily life movements), and (2) CBT-I (i.e., sleep psychoeducation, behavioral and cognitive therapy, correcting sleep hygiene, and relaxation therapy) can be deployed for the management of patients suffering from chronic spinal pain.

Impact statement:

Because of the major impact sleep disturbances have on pain and disability, insomnia as a comorbidity should no longer be ignored when treating patients with chronic spinal pain.
The underlying pathophysiology of nCSP points toward alterations in the central nervous system, making it a complex and multidimensional sensation involving also psychological factors. Accordingly, treatments targeting local structures appear to have little effect in patients with nCSP. In contrast, contemporary cognitive-behavioral therapies (CBT) for nCSP, consisting of pain neuroscience education and cognition-targeted exercise therapy (CTET), among others, yields promising results. However, potentially associated comorbidities such as chronic insomnia generally remain untreated in physical therapy practice. Ignoring sleep complaints, though, might delay or impede effective pain management in a subgroup of nCSP patients with comorbid insomnia given that disturbed sleep increases pain sensitivity. Moreover, a good night’s sleep seems to increase daytime physical activity which may contribute to tackling nCSP. These factors emphasize the value of addressing sleep within therapy.

Chronic insomnia disorder is defined as unsatisfactory sleep, often associated with difficulties initiating and/or maintaining sleep in the presence of adequate opportunity and circumstances to sleep, and with consequences on daytime functioning. Additionally, sleep problems must be present more than 3 days a week for at least 3 months. Percentages reflecting the prevalence of clinical insomnia as a comorbidity in chronic pain conditions range from 53% to 90%. Also in persons reporting spinal pain, sleep problems are dominantly present with reported rates of 50% and more. In practice, therapy for chronic insomnia is often limited to pharmacological interventions and/or sleep education. However, in addition to various adverse effects, evidence for the effectiveness of sleep medication appears limited, especially in the long-term. Furthermore, sleep hygiene as a stand-alone therapy, containing advice on behavioral and environmental factors, yields unconvincing results as well. The recommended first-line therapy for chronic insomnia is therefore cognitive-
behavior therapy for insomnia (CBT-I). Indeed, this multicomponent treatment approach, encompassing sleep psychoeducation, time-in-bed restriction, cognitive and behavioral therapy (stimulus control) and relaxation training, appears to have manageable adverse effects and has been found to be effective in the long-term treatment of chronic insomnia.

Application of CBT-I in People with Chronic Pain and Comorbid Insomnia

Various randomized controlled trials (RCTs) established the effectiveness of non-pharmacological sleep interventions (e.g., sleep hygiene, CBT-I) to enhance sleep outcomes in various chronic pain populations, but fail to demonstrate a consistent impact on pain. Moreover, the current lack of a holistic approach may increase the reliance to polypharmacy with potential risks of side effects and an amplification of the comorbidity. Therefore, the integration of a hybrid therapy, targeting both insomnia and chronic pain, seems appropriate to optimize nCSP management. Indeed, such an integrated approach, aiming at simultaneously addressing the physiological, psychological, and behavioral aspects of the coexisting complaints, could break the vicious cycle as improvement in one disorder might facilitate progress in the other. A long-term view in interpreting treatment effects is crucial in this context, given the essential but sometimes time-consuming role of cognitive and behavioral changes as of function of lifestyle factors.

Rationale behind a Hybrid Therapy Approach

Application of a Hybrid Therapy Approach in People with Chronic Pain and Comorbid Insomnia
Two pilot studies in a chronic pain population demonstrated the effectiveness of a therapy approach that combined CBT for sleep and pain on both sleep and pain-related parameters compared to waitlist controls.\(^{33,34}\) Additionally, findings from one RCT in elderly patients suffering from osteoarthritis supports the hypothesis that sleep management may beneficially impact pain and quality of life.\(^{35,36}\) Together, these findings provide a robust proof of concept to integrate a blended treatment approach in the management of patients with nCSP and comorbid insomnia, combining CBT-I and PNE with more active treatment modalities.\(^{37}\)

**Bidirectional relationship between sleep and pain**

While experimental and clinical studies demonstrate a bidirectional relation between central sensitization and sleep disturbances, accumulating evidence suggests that disturbed sleep is a more reliable predictor for pain development than the other way around.\(^{38,39}\) Consequently, in addition to being a perpetuating and intensifying factor for pain, insomnia also appears to be an etiological determinant.\(^{12}\) Thus, a vicious cycle is created in which sleep problems enhance pain sensitivity, leading to generalized hyperalgesia, which further results in an increased incidence or severity of sleep disturbances.\(^{33,40}\) Typically, however, sleep impairments and chronic pain are considered symptoms and consequently treated independently, disregarding possible common underlying interacting mechanisms.

**Potential underlying mechanisms**

The vital yet extremely complex homeostatic processes of both sleep and central sensitization implicate the involvement of many intricate, dynamic and multifaceted interactions. Numerous mechanisms have already been suggested to explain the association between sleep and chronic pain however, the exact underlying mechanisms of this interaction have not yet been elucidated.\(^{12,13,41,42}\)
The balance of the immune system and the interrelated hypothalamus-pituitary-adrenal (HPA) axis is thought to be affected by disturbed sleep, shifting to a potentially glia-mediated pro-inflammatory state associated with stress and anxiety.\(^\text{12,13}\) This implies increased levels of pronociceptive cytokines, prostaglandins and nitric oxide contributing to a heightened pain sensitivity, characteristic of individuals with chronic pain.\(^\text{43}\) Also, increased adenosinergic activity at A\(_{2A}\) receptors is suggested to affect nociceptive processing leading to hyperalgesia.\(^\text{13}\)

Furthermore, several monoaminergic systems appear to be involved in the altered pain modulation associated with sleep disturbance.\(^\text{13,41}\) Thus, a potential role is suggested for dopamine, a wake-promoting mediator, key neurotransmitter of the reward system and involved in endogenous analgesia. Increased pain sensitivity due to sleep deprivation may thereby be associated with lowered dopaminergic activity.\(^\text{13,41}\) Also, dysfunctional serotoninergic pathways, involved in both the sleep-wake system and endogenous pain modulation, could mediate the hyperalgesic effects following disrupted sleep.\(^\text{12,13}\) The orexinergic system and its related neuropeptides (orexin A and B) are involved in both the sleep-wake process and nociceptive transmission and modulation. It is therefore hypothesized that alterations in this system have a role in hyperalgesia resulting from poor sleep.\(^\text{13}\)

Furthermore, a decrease in available opioid receptors, which play a principal role in antinociceptive central pain modulation, as a consequence of sleep disturbances is suggested resulting in less responsivity to opioids and thus reduced pain inhibition, thereby facilitating pain in chronic diseases.\(^\text{12,13}\)

The neurohormone melatonin not only has a sleep-promoting effect controlled by light exposure, but it also has an analgesic and anti-inflammatory function, potentially providing a role in the relationship between sleep and pain.\(^\text{13}\)
In general, disrupted sleep causes increased sensitivity and responses to nociceptive stimuli, and thus exacerbates existing pain hypersensitivity, reducing treatment effectiveness and delaying recovery. Despite the fact that the results of large RCTs are still pending, implementing sleep management in physical therapy should be at the top of the research agenda given the major impact of sleep disorders on chronic pain and the high degree of comorbidity of these disorders. The purpose of this paper, therefore, is to provide practical guidance on the application of current evidence-based knowledge on the association between sleeplessness and chronic spinal pain.

**Hybrid Therapy Approach in People with nCSP and Comorbid Insomnia**

Before applying the hybrid CBT approach, a thorough screening of the patient should be completed in order to exclude underlying sleep (e.g., obstructive sleep apnea, restless legs syndrome) or psychological (e.g., depression) pathologies. Self-reported questionnaires (e.g., Insomnia Severity Index, Pittsburgh Sleep Quality Index, Epworth Sleepiness Scale, Brugmann Fatigue Scale) can be used, and if indicated, referral for a polysomnography should be considered. Screening techniques and the content of the anamnesis to determine the presence of insomnia are beyond the scope of this paper but have been previously described.

Communication is a crucial cornerstone throughout therapy, with the physical therapist implementing techniques of motivational interviewing aimed at developing and engaging the patient’s autonomy with respect to behavioral change. This process of change is linked to the transtheoretical model of change in which six phases are dynamically passed through (i.e., precontemplation, contemplation, action, maintenance, relapse and termination). Further
underlying theoretical frameworks consists of various concepts such as the social cognitive theory, self-determination theory and self-regulatory model, all of which share common constructs. As such, the expectations of the patient about the implications of behavior change, the patient’s perception of his or her self-efficacy (i.e., patient’s belief in his or her own ability to change behavior) and the social context of the behavior are all addressed in these theories. A Socratic communication method (i.e., open questions, empathic and reflective listening) is applied to assess treatment acceptance, strengthen the patient-therapist relationship and resolve ambivalence, allowing the individual’s intrinsic motivation to be identified and reinforced. This way, therapeutic alliance and adherence are facilitated, with the patient adopting an active role during therapy. However, in contrast to finding positive outcomes in patients with low back pain resulting from a strong therapeutic alliance, evidence that improved adherence in the short-term also affects therapy outcomes is lacking to date.

An example of the practical implementation of the hybrid therapy approach applied to a case can be found in the supplementary material.

1. General Education

Pain Neuroscience Education

Pain neuroscience education (PNE) entails the reconceptualization of pain to alter patients’ dysfunctional pain cognitions and disease perceptions in order to enable the application of active exercise therapy in a further therapy stage.
In an initial educational session, the purpose and course of acute pain in the nervous system as well as the changes that occur in chronic pain, including the mechanisms involved in central sensitization, are discussed based on a visual presentation (e.g., http://www.paininmotion.be/education/tools-for-clinical-practice).\textsuperscript{58,60} The content of the presentation intends to change the individual's perception that pain is related and proportional to tissue damage. Through the acquired knowledge, pain should be disconnected from tissue damage by associating it primarily with altered processes in the brain, making it less threatening.\textsuperscript{61} After this educational session, patients receive a booklet and/or online module containing the taught material (e.g., http://www.paininmotion.be/education/tools-for-clinical-practice). Patients are asked to review this information at home to promote in-depth learning. In a subsequent session, questions are asked regarding the individual’s experiences and perceptions of pain (e.g., by using the self-reported Pain Catastrophizing Scale) to assess the patient’s is preparedness to move to a next therapy stage.\textsuperscript{62} The subject’s responses are then discussed and misinterpretations are corrected to ensure that the information received about pain is optimally implemented during CTET.

The effectiveness of PNE in improving both physical and psychological factors in various chronic pain conditions has been described previously.\textsuperscript{60,63} The combination with a physical intervention (e.g., exercise therapy) is recommended to optimize the synergetic treatment outcome.\textsuperscript{7,60,64,65}

**Sleep Psychoeducation**

The main objectives of sleep psychoeducation are to inform, correct patients’ dysfunctional beliefs and obtain realistic expectations regarding sleep and its relationship to pain so that therapy components can be applied and therapy adherence is encouraged. Thus, it provides
patients with a solid and science-based understanding of natural sleep and the underlying psychological and behavioral factors in the development of chronic insomnia. In one session, basic concepts of normal sleep and its corresponding sleep mechanisms are explained using an illustrated presentation. Appendices 1 to 4 detail the sleep principles essential within sleep psychoeducation.

2. Behavioral Therapy

Time-in-Bed Restriction Therapy

Sleep Diary

To obtain a subjective estimate of an individual’s sleep duration, waking periods during the night, and the timing of these parameters relative to a 24-hour period, a sleep diary is utilized during an initial observation phase. The patient is instructed to complete the sleep diary during an initial week without making any changes to the actual sleep pattern, other than avoiding clock-watching, to obtain an accurate baseline for applying further therapy. Indeed, a sleep diary is a valuable tool for a physical therapist to evaluate self-reported insomnia based on sleep efficiency (SE; proportion of time in bed spent asleep), sleep quality, feelings upon waking and throughout the day. Additional information on physical activity and the intake of medication, alcohol, nicotine, and caffeine could also be noted in the diary, as these are important factors affecting sleep (see also sleep hygiene). The patient is additionally asked to indicate the time points when fatigue or sleepiness occurred. To be complete, information about experiencing pain may be recorded in the sleep diary. To obtain complete information on relevant sleep aspects, discussion of the sleep diary with the patient is appropriate to further nuance the sleep and wake experiences.
A panel of experts has developed a standardized sleep diary (Consensus Sleep Diary, CSD, https://consensussleepdiary.com/) of which the psychometric properties support validity, clinical utility and usability. Alternative formats of sleep diaries are used as well, including time charts, clock faces, or electronic diaries. Figure 1 shows an example of a blank sleep diary based on a time chart that can be used in practice. Instructions for completing this sleep diary can be found in Box 1.

**Interpretation of the Score Form**

The sleep diary is scored together with the patient during a subsequent session by calculating the average values of the various sleep parameters using a score form. Figure 2 shows an example of a blank score form and Box 2 provides the instructions for calculating the various sleep parameters. Both the patient and physical therapist thus gains insight into the patient's subjective sleep pattern, providing a broader perspective and subsequently specific behavioral changes can be targeted. The sleep diary and its accompanying score form are therefore used as data source on a week-to-week basis throughout further therapy for two reasons: (1) to monitor time-in-bed restriction, and (2) to observe treatment-related changes in sleep outcome parameters.

**Time-in-bed restriction**

Time-in-bed restriction aims to reduce time spent awake in bed, achieve a state of mild sleep deprivation by initially restricting the time spent in bed to the average total sleep time, thus increasing the homeostatic sleep pressure, and realigning the circadian rhythm to this process (see Appendix 2). This objective is reached by altering perpetuating coping strategies, thereby improving sleep latency and general sleep quality.\(^{68}\)
By scoring the sleep diary, SE is calculated which represents the possible mismatch between time spent in bed and total sleep time, providing the primary indicator for the application of time-in-bed restriction. Based on the SE percentage, an appropriate sleep window (i.e., target amount of sleep opportunity in bed) is defined according to the average total sleep time for the past week. First, the hour of awakening is determined and then a target time for going to bed is obtained by backward counting the number of hours of effective sleep from the time of awakening in the morning (e.g., a sleep window of 7 hours and 30 minutes could result in a total time in bed from 11.00 pm to 6.30 am).

The following instructions are proposed concerning SE: (1) if SE is < 85%, a new sleep window is determined by making the total time in bed equal to the mean total sleep time of the past week (sleep window ≥ 5 hours because of safety and health concerns), (2) if SE is 85 - 90%, the sleep window is retained; and (3) if SE is > 90%, the sleep window is extended with 15 minutes, allowing for a potential increase of total sleep time. So, according to these guidelines, a time-in-bed restriction is obtained if SE is < 85%.

As time-in-bed restriction can result in mild sleep deprivation, and consequently daytime sleepiness, it is important to explain the purposes of sleep-wake scheduling (reducing the amount of awake time spent in bed rather than shortening sleep duration; increasing sleep propensity) to the subject. In addition, the physical therapist should evaluate the individual’s intrinsic motivation by Socratic communication and establish a trusting therapeutic relation before implementing time-in-bed restriction in therapy. As a consequence of sleep deprivation, daytime sleepiness may be provoked, which can indicate the therapy effectiveness as it reflects increased sleep propensity. However, the enhanced sleepiness
seem to occur in the first weeks after the introduction of the time in bed restriction and usually returns to a baseline level thereafter.\textsuperscript{70}

During further therapy, each session starts with reviewing the sleep diary, interpreting SE and repeating the procedure of time-in-bed restriction by altering the sleep window if necessary. Indeed, as SE increases over time, the sleep window is gradually extended, allowing an increase in total sleep time until an optimal equilibrium is found between sleep duration and efficiency, with possible beneficial effect on pain parameters.

\textit{Sleep compression}

Sleep compression is a milder alternative to time-in-bed restriction since sleep opportunity is gradually reduced over several weeks rather than radically curtailing sleep early in therapy.\textsuperscript{69,71} Compression of time in bed is attained by either advancing wake-up time or delaying bedtime until the average baseline sleep time is reached. This strategy may be applied in anxious patients and individuals in whom time-in-bed restriction is not tolerated because of the abrupt decrease in sleep opportunity. However, sleep compression also entails some disadvantages as it may take longer for the insomnia symptoms to subside, possibly resulting in frustration and/or a lack of therapy adherence. More specifically for nCSP patients, this approach may be appropriate to prevent an increase in pain sensitivity given the hyperalgesic response caused by sleep deprivation.\textsuperscript{71}

\textbf{Medication}

\textit{Analgesics}

Sleep disturbance negatively affect the efficacy of pain medications (e.g., opioids), increasing the risk of dose escalation and consequently dependence.\textsuperscript{41} In patients with chronic pain, it is
recommended that the dosage and timing of intake of analgesics should be determined and adjusted in consultation with a physician to minimizing its sleep-disrupting effects.\(^{13}\)

**Sedative Hypnotics**

Tapering and subsequently discontinuing the intake of sleep medications are among the therapy objectives. Indeed, the risk of becoming dependent on hypnotics increases with frequent and prolonged use. Before starting to withdraw the medication, the therapist should check whether the patient is motivated to stop the intake of medication. Since there are risks for adverse effects such as rebound insomnia, if sleep medication intake is stopped suddenly and without medical supervision, it should be phased out gradually.\(^{72}\) For this reason, it is necessary to collaborate with the prescribing physician to determine if and how sleep medication can be phased out. If the patient takes a combination of different sleep agents, the physician decides which medication will be discontinued first. A gradual tapering schedule is than created in consultation with both the physician and the patient, consisting of a 25% reduction in medication, at intervals of 1 to 2 weeks, until the lowest available dose is reached.\(^{72}\)

**Stimulus Control**

Stimulus control includes behavioral instructions, based on classical and operant conditioning that counteract inconsistent pain and sleep behavior.\(^{73,74}\)

**Sleep**

Patients with chronic insomnia often develop a classically conditioned response, namely between the bed(room) and lying awake. Therefore, stimulus control regarding sleep aims to restore the positive association between the bed(room) and sleep and to (re)establish a
Specific sleep related stimulus control instructions to the patients are the following: (1) only go to bed if you feel sleepy, (2) get out of bed when unable to sleep within 15 minutes, (3) use your bed only for sleeping and sexual activities, (4) avoid naps during the day, and (5) maintain a consistent sleep-wake schedule.

**Pain**

Specific to nCSP, inactivity and inappropriate protective or safety behaviors (e.g., adjusting or avoiding bending forward because it provoked pain in the past; co-contracting stabilizing muscles to protect the spine) may be applied as coping strategies and need to be targeted during exercise therapy (see CTET). In addition, pain and its associated reactions may indirectly contribute to sleep issues. Nighttime physical discomfort, such as spinal pain, can interfere with sleep leading to a loss of control, pain catastrophizing and consequently pre-sleep cognitive hypervigilance. Extending time in bed or resting in bed during the day as a function of pain or fatigue rather than effective sleepiness, may also reduce SE and relegate the bed(room) to a weaker cue for sleep in patients with CSP. Addressing these issues during therapy is indicated.

**3. Cognitive Therapy**

The aim of cognitive therapy is to change an individual’s dysfunctional beliefs and expectations about sleep and/or pain by verbal interventions and behavioral experiments. Modifying these perceptions reduces emotional and somatic arousal, addresses barriers for the required behavioral change, and may subsequently improve sleep and/or pain parameters, thus breaking the vicious cycle between arousal and sleep problems and/or pain. The therapist guides patients to reevaluate their thinking by providing key messages (e.g., ‘insomnia is not
the (only) cause of all daytime dysfunctions, there may be other explanations for these
deficiencies') and behavioral experiments in the form of homework (e.g., testing out the effect
of active versus passive behavior as a function of pain). Thus, a more realistic and rational view
of sleep and/or pain is achieved. In addition, problem-solving training, such as noting
problems and the associated first step of the solution before bedtime, and acceptance-based
therapies can reduce arousal.

General education (PNE and sleep psychoeducation) constitutes an essential part of the
cognitive component within therapy and aims to facilitate the implementation of more active
interventions driven by patient self-efficacy. During the initial therapy phase, education is
provided that serves as the foundation for the subsequent treatment progression, turning it
into a continuous process that is referred to repeatedly.78,79

In the context of CTET, pain education ensures the ability to apply a time-contingent rather
than a pain-contingent approach. This involves performing an exercise to the frequency and
intensity agreed upon during therapy, without modifying or stopping when experiencing
pain.80

Insomnia and spinal pain are both more prone to evolve into chronic diseases when an
individual perceives this complaints as a loss of control and is concerned about the
consequences.78,81 Indeed, dysfunctional cognitive responses feed the vicious cycle of
insomnia and pain.78 Increased emotional stress produces a state of both emotional and
physiological hypervigilance, possibly enhanced by (presleep) somatic arousal caused by pain,
which all adversely affects sleep.82 An essential objective of cognitive therapy is therefore to
enhance the feeling of control and to strengthen coping skills when dealing with sleep
problems and their consequences.
Sleep hygiene encompasses a range of advice related to lifestyle (e.g., physical activity, diet, substance use) and environmental factors (e.g., temperature, noise, light) that, although often not the primary cause of chronic insomnia, may contribute to the perpetuation of sleep problems. Educating the patient about the relative risks of these factors can help improve treatment progress. Thus, the purpose of sleep hygiene instructions is to tackle unfavorable sleep behaviors by providing appropriate information to improve sleep quality. Box 3 shows the advices provided to the patient regarding sleep hygiene. Some of these factors overlap with the components of time-in-bed restriction and stimulus control.

Caffeine consumption and smoking of nicotine are detrimental to sleep since both stimulate the central nervous system. Alcohol, in turn, is a suppressor of the central nervous system resulting in a short sleep onset latency, but reduced sleep duration and poorer sleep quality due to withdrawal symptoms. Hence, the use of these substances before bedtime is discouraged. Additionally, intensive exercise within an hour before bedtime is not recommended because it activates the autonomic nervous system and increases body temperature, leading to a delay in the onset of sleep. However, energy expenditure from physical activity does promote sleep when exercise is performed at other times of the day and is also beneficial in reducing stress.

4. Relaxation training

(Hyper)arousal is known as one of the factors that contributes to the onset and maintenance of insomnia. In turn, poor sleep can reduce a person's ability to cope with everyday stressors. Relaxation therapy therefore aims to reduce somatic and/or cognitive arousal and
its associated autonomic stress response in function of indirectly improving both sleep quality
and pain. Practicing relaxation techniques before bedtime is encouraged in the presence of
pre-sleep arousal, where the deactivation of the stress system through relaxation can be
further complemented by adapting evening activities and implementing breaks (i.e., mental
or physical rest) throughout the day. Learning a relaxation technique is a time-consuming,
progressive process that requires daily practicing for a period of a few weeks. The selection
of the applied relaxation technique depends on the presence of cognitive or somatic arousal.
In order to reduce racing thoughts (i.e., cognitive arousal), attention-oriented techniques as
visualization or thought stopping techniques are appropriate to focus on more pleasant
images or thoughts. In the case of dominant muscle tension, progressive muscle relaxation
is indicated, whereby certain muscle groups are alternately contracted and relaxed. Control
of physiological parameters through auditory or visual feedback, referred to as biofeedback,
is another therapy that can be recommended in somatic arousal. The choice of relaxation
technique is further determined by the patient’s preference. Previous positive or negative
experiences with relaxation may additionally influence patient’s perspectives, so questioning
and considering these factors is critical. If stress has a profound impact on the individual, more
comprehensive stress management offers a valuable complement to relaxation therapy.
More detailed information on relaxation and stress management can be found elsewhere.

5. Cognition-targeted exercise therapy

Once a person suffering from CSP has adopted the biopsychosocial principles of the PNE, CTET
can be initiated aiming to alter the patient’s pain memory in the brain. The amygdala as part
of the pain neuromatrix is a key brain area involved in the memory of painful movements. The
overactivation of the pain neuromatrix due to neuroplasticity, results to maladaptive changes in response to stimuli. By cooperating with other brain regions such as the anterior cingulate cortex and hippocampus, pain memory directs protective safety behaviors in response to movements previously experienced as painful. As such, this long-term pain memory can interpret certain daily life movements as threatening, causing pain and resulting in an inappropriate safety response (e.g., antalgic postures, co-contraction of stabilizing muscles) that interferes with a natural and functional way of moving. Cognition-targeted exercise therapy therefore aims to systematically desensitize the central nervous system by substituting the dysfunctional pain memory through enhancing pain-coping strategies via repeated and progressive exposure to certain challenging movements or postures. Again, appropriate communication forms a crucial therapy component whereby the cognitions, perceptions and expectations of the patient regarding a specific movement and the possible associated pain are constantly questioned. So, based on the patient’s individual fear of movement, a time-contingent, individual approach is applied whereby the degree of challenge of the exercises is built up progressively, starting from simple analytical to more dynamic and functional movements. The patient is also encouraged to do daily home exercises to further alter the maladaptive pain memory. Examples of cognition-targeted exercises and concurrent communication were described elsewhere. Therapeutic effects of exercise training appear to be manifested centrally, including psychological, neurophysiological and cognitive modifications, rather than being directly attributed to the rectification of a biomedical deficiency of the musculoskeletal system. Thus, the outcome of motor control training in both neck and back pain appears not to be superior compared to other types of exercise. Instead, motor control training might be counterproductive by encouraging improper safety behavior, increasing fear of movement,
and consequently pain when performing a movement without co-contraction of the stabilizing
muscles. A9,95

6. Relapse prevention

Relapse prevention seeks to avoid long-term recurrence of symptoms by reiterating the
therapy principles and identifying potential risk factors or situations.26 It thus assumes the
facilitation of the further development or maintenance of the behavioral change brought
about as a function of more appropriate coping strategies.96 If necessary, the behavior change
theories can be invoked again in order to prevent further relapse. The therapist evaluates
whether the patient’s objectives regarding sleep and pain, set in the first session, are achieved,
and if not, what could be the underlying causes. Furthermore, the patient is advised to practice
the acquired competencies to further optimize pain parameters and sleep (and wake) quality.

With respect to chronic spinal pain, relevant home exercises and possible paths for further
progression are discussed. The emphasis during the sleep evaluation is on the progress in
general without focusing on a bad night or week. Thus, more than achieving good sleep
parameters and scores, sleep therapy aims to improve patient’s sleep perception by accepting
that sleep (and wake) may not be perfect on a daily basis, resulting in less concern and anxiety
about sleep. Booster sessions (e.g., in person, via telephone) may be appropriate to prevent
relapse.

How to put Hybrid Therapy into Practice?

Typically, CBT-I is delivered by psychologists or health providers with training in sleep medicine
and behavioral and/or cognitive therapy.69 However, to date there is a lack of specialists who
can accurately treat the vast number of patients with insomnia.\textsuperscript{97} Considering up to 90% of patients with chronic pain exhibit impaired sleep,\textsuperscript{18,19} providing CBT-I within the physical therapy practice may be a beneficial addition to compensate for this deficit in sleep specialists. Although many physical therapists consider sleep to be an important factor likely to affect therapy outcomes, a lack of knowledge on this topic seems to account for the fact that sleep is not yet adequately addressed in daily physical therapy practice.\textsuperscript{98} Integrating information on sleep into the entry-level curriculum of physical therapy students and providing continuing training opportunities to practicing physical therapists appears to be a crucial step in optimizing treatment regarding insomnia.\textsuperscript{97} As one could argue the implementation of CBT-I in physical therapy, a recent study has established that physical therapists provided with specific training (e.g., on behavior change theories) are able to effectively carry out cognitive behavioral therapy in clinical practice.\textsuperscript{37,97} In the future, the introduction of a certificate of competence for CBT-I specialists, as an extension of a clinician’s practice license, may be a useful option for ensuring the quality of therapy.\textsuperscript{99} However, proper billing and reimbursement of CBT-I still appears to pose problems for physical therapists. In contrast, clinicians practicing in the mental health field, providing cognitive and/or behavioral treatments, are familiar with and accustomed to billing that is consistent with mental health rates and co-payments.\textsuperscript{97} As the implementation of CBT-I in the health care systems seems to vary across the world,\textsuperscript{97,99} a need exists for more accessible, multidisciplinary incorporation of CBT-I within all health care systems.

Based on the dominant complaint and the patient’s history, the focus within therapy may be on sleep or pain. However, the concept of simultaneously addressing sleep and pain, by alternating exercise with sleep therapy sessions, remains the starting point of the approach. Furthermore, the practical implementation of the various therapy components should be
tailored to the patient and the possibilities within the therapeutic setting. Thus, the format of
the sessions may vary for cost-effectiveness, providing individual, group sessions, or a
combination of both. Additionally, group sessions promote interaction compared to an
individual approach. The option of online sessions rather than live sessions may also offer a
solution regarding cost-effectiveness. Dose and duration of therapy sessions vary across
literature. Previous findings indicated that 4 or more individual CBT-I sessions are more
effective in treating insomnia compared to fewer sessions or self-help interventions. The
duration of a session may range between 30 minutes and 2 hours. Table 1 presents a proposal
of an alternating therapy structure between sleep- and pain-focused sessions spread over 14
weeks. Figure 3 shows an example of the substantive organization of the various CBT-I
components over 6 sessions.

Contra-indications for the application of CBT-I

A number of contra-indications are kept in mind to use CBT-I within nCSP management. In the
presence of a sleep pathology, a severe addiction or mental disorder that has not yet been
treated, the absence of negative conditioning about sleep, and insufficient motivation for
behavioral change are considered contraindications for applying CBT-I. Adequate screening
and referral if required are indicated to ensure optimal therapy for the patient. Yet, providing
sleep education and hygiene in this population remains recommended even in case of these
contra-indications.

Conclusion
Growing evidence for the association between sleep and pain indicates that this comorbidity should no longer be ignored in the treatment of patients with chronic spinal pain. A therapy approach in which both chronic spinal pain and sleep are addressed seems appropriate. To meet this challenge, the current evidence-based contemporary neuroscience approach consisting of PNE and CTET is combined with CBT-I in order to target pain, disability and sleep complaints.

**Author Contributions**


Final approval before submission: B. Cagnie, M. Meeus, O. Mairesse.

**Role of the Funding Source**

This paper was funded by the Applied Biomedical Research Program, Research Foundation Flanders (Fonds voor Wetenschappelijk Onderzoek Vlaanderen), Belgium (FWO-TBM project no. T001117N). The funder played no role in the design, conduct, or reporting of this study.

**Conflict of interest**
O. Mairesse is involved in the training of CBT-I for psychologists at the University Hospital of Antwerp organized, by the Belgian Association for Sleep research and Sleep medicine (BASS).

J. Nijs authored a Dutch book on PNE.
References


8. Moseley GL. Joining Forces – Combining Cognition-Targeted Motor Control Training with Group or Individual Pain Physiology Education: A Successful Treatment For


594 17. Riemann D, Baglioni C, Bassetti C, et al. European guideline for the diagnosis and


34. Tang NKY, Goodchild CE, Salkovskis PM. Hybrid cognitive-behaviour therapy for individuals with insomnia and chronic pain: A pilot randomised controlled trial. *Behav*


77. Morgenthaler T, Kramer M, Alessi C, et al. Practice Parameters for the Psychological
757 and Behavioral Treatment of Insomnia: An Update. An American Academy of Sleep
759
761
763
765
767
769
771
773
775


Table 1. Practical organization of therapy sessions over a treatment period of 14 weeks

<table>
<thead>
<tr>
<th>WEEK</th>
<th>THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2</td>
<td>PNE</td>
</tr>
<tr>
<td>3</td>
<td>CBT-I</td>
</tr>
<tr>
<td>4</td>
<td>CBT-I</td>
</tr>
<tr>
<td>5</td>
<td>CBT-I</td>
</tr>
<tr>
<td>6</td>
<td>CBT-I</td>
</tr>
<tr>
<td>7</td>
<td>CTET</td>
</tr>
<tr>
<td>8</td>
<td>CBT-I</td>
</tr>
<tr>
<td>9</td>
<td>CTET</td>
</tr>
<tr>
<td>10</td>
<td>CBT-I</td>
</tr>
<tr>
<td>11-14</td>
<td>CTET</td>
</tr>
</tbody>
</table>

PNE, pain neuroscience education (group session + online session + individual session); CTET, cognition targeted exercise therapy; CBT-I, cognitive behavioral therapy for insomnia.
Figure 1. Sleep diary

![Sleep Diary Image]

Figure 2. Score form

![Score Form Image]
Figure 3. Timeline for applying cognitive behavioral therapy for insomnia

<table>
<thead>
<tr>
<th>Week from</th>
<th>Monday - Tuesday</th>
<th>Tuesday - Wednesday</th>
<th>Wednesday - Thursday</th>
<th>Thursday - Friday</th>
<th>Friday</th>
<th>Saturday</th>
<th>Sunday - Sunday</th>
<th>Sunday - Monday</th>
<th>MEAN of the week</th>
<th>8 hours (mean/SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time in bed (TIB)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep onset latency (SOL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wake after sleep onset (WASO)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total wake time (TWT = SOL + WASO)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total sleep time (TST = TIB - TWT)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep efficiency (%) (TST/TIB x 100)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep quality (1/30)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daytime napping (1/30)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Main components per session:

- Session 1 - sleep psychoeducation + explanation on how to fill in the sleep diary
- Session 2 - explanation on how to score the sleep diary and interpret the score form + time-in-bed restriction
- Session 3 - relaxation training
- Session 4 - relapse prevention
- Session 5 - cognitive therapy
- Session 6 - stimulus control
- Session 7 - behavior therapy
- Session 8 - medication
Session 3 - reevaluation of sleep window + possible extension/reduction of sleep window + stimulus control

Session 4 - reevaluation of sleep window + possible extension/reduction of sleep window + sleep hygiene

Session 5 - reevaluation of sleep window + possible extension/reduction of sleep window + relaxation training

Session 6 - evaluation + relapse prevention

Box 1. Instructions on how to fill in a sleep diary

- Complete the items about the previous night in the sleep diary at a set time in the morning, about 20-30 minutes after waking to limit the influence of sleep inertia (i.e., the period of reduced alertness and/or performance after waking up).

- In the evening, enter in the sleep diary any notable additional items that occurred during the day (i.e., intake of alcohol, caffeine, napping, etc.).

- Put your sleep diary in a visible place to reduce the chance of forgetting to fill it out. If you do forget, leave the boxes for that night open rather than filling them in later.

- Do not check the clock during the night. The sleep diary is an estimate of your sleep and therefore subjective.
• Each hour is divided into 4 boxes, each representing 15 minutes. All times requested should be rounded according to these 15-minutes boxes.

• For the time you go to sleep and turn off the light, put a downward arrow (↓) on the dividing line between the 2 boxes for the corresponding hour.

• For the time you get up in the morning, put an upward arrow (↑) on the dividing line between the 2 boxes for the corresponding hour.

• If you read, work or watch television in bed during the evening, indicate this with a vertical line at the time you start doing so (followed by the downward arrow when you turn off the light and try to sleep).

• Color all boxes for the hours you slept (according to your feeling) at night or during the day (i.e., napping).

• Arce the boxes corresponding to the time in bed without sleeping (e.g., if you spent 15 minutes awake in bed, arce the corresponding box).

• Mark the intake of medication (M) caffeine (C), alcohol (A), or nicotine (N) and physical activity (P) in the corresponding boxes. Further specifications can be given under ‘treatment and remarks’ (e.g., name and dose of medication).

• Mark the times when you felt sleepy (S; subjective experience of a need for sleep and an effort to stay awake, resolved by sleep) or fatigued (F; feeling of exhaustion, often caused by muscular, emotional or mental exertion and elevated by rest) in the corresponding boxes.

• Sleep disturbing factors (e.g., pain, environmental noise) may be additionally noted under ‘treatment and remarks’. Other notable issues (e.g., intense pain, vacation) may also be listed under this heading.
• **Time in bed (TIB):** count the number of boxes of 15 minutes between going to bed (↓) and getting up (↑). Time out of bed during the night is not counted.

• **Sleep onset latency (SOL):** count the number of shaded boxes of 15 minutes between going to bed (↓) and the first colored box.

• **Wake after sleep onset (WASO):** count the number of shaded boxes between the first colored box and getting up in the morning (↑).

• **Total wake time (TWT):** sum up the SOL and WASO.

• **Total sleep time (TST):** subtract the TIB from the TWT.

• **Sleep efficiency (SE):** divide the TST by the TIB and multiply by 100 to obtain a

• Rate your sleep quality and rested feeling (awakening quality) from 0 to 10 (0 = not good, 10 = very good) at the time you complete the sleep diary.

• Rate your feelings during the past day from 0 to 10 (0 = not good, 10 = very good) in the evening.

**Box 2. Instructions on how to calculate sleep parameters of score form**
Box 3. Sleep Hygiene Instructions

- Avoid caffeine consumption 4 to 6 hours before bedtime.
- Do not smoke before going to bed or when you wake up at night.
- Avoid alcohol consumption 4 to 6 hours before bedtime.
- Avoid heavy meals before bedtime.
- Avoid intense training 2 hours before bedtime.
- Provide comfort to your bedroom.
- Avoid extreme temperatures in your bedroom.
- Keep your bedroom quiet and dark.
- Avoid active interacting with screens 1 hour before bedtime.

percentage, i.e., \((\text{TST} / \text{TIB}) \times 100\).

- Calculate the average for the week of each sleep parameter by dividing the sum of every row by 7.
- Because each box represents 15 minutes, divide the number of boxes by 4 to obtain the number of hours. The number before the comma indicates the number of hours, the number after the comma indicates the number of quarters in decimal places (i.e. 0.25 = 15 minutes, 0.5 = 30 minutes and 0.75 = 45 minutes).
APPENDIX

Appendix 1. Hypnogram

The stable sequence of the different sleep cycles (x-axis) and stages (y-axis) of a healthy night’s sleep are represented in a hypnogram, reflecting an individual’s personal sleep architecture. The time span of one cycle typically ranges from 90 to 120 minutes, resulting in a number of...
4 to 6 cycles per night. During one cycle, an individual passes through the various sleep stages. Thus, in a first non-rapid-eye movements (REM) phase (N1), the transition from wake to sleep occurs. It concerns a light sleep that lasts only a few minutes before progressing to the next stage, the second non-REM phase (N2). During this phase muscle tension will continue to decrease. In addition, heart rate and respiration also slow down in preparation for deep sleep (N3) in which a low frequency will eventually be reached. Finally, during REM sleep, heart rate and respiration become irregular. Furthermore, considerable brain activity occurs whereby information and experiences of the past day are ordered and processed. A shift in sleep phases during the night can be observed, i.e., a dominant slow wave sleep (N3) at the beginning of the night developing into more dominant REM sleep by morning. Moreover, this figure also shows that short awakenings throughout the night are a natural phenomenon and usually occur unconsciously due to amnesia during the night.

Appendix 2. Two-process model of sleep regulation adapted from Borbely 1982

The graph shows the two-process model of sleep regulation representing the timing of the sleep-wake alternation resulting from two interacting processes: a homeostatic process (process S) that reflects the sleep-dependent accumulation of sleep pressure, and an
endogenous sleep-independent circadian rhythm (process C) tracking the environmental time. Process S thus regulates the sleep depth and intensity characterized by a monotonous increase in sleep propensity during wake and a subsequent exponential decline during sleep. Taking a daytime nap consequently reduces sleep pressure, which is likely to lead to problems initiating sleep around bedtime and lighter sleep during the night. Sleep deprivation, on the other hand, results in a buildup of sleep homeostasis, causing sleep debt. Sleep homeostasis can be optimized by going to bed only when sleep pressure is sufficiently present. As a function of this, time in bed restriction may be appropriate, as this will minimize waking time in bed and thus delay the time to sleep. Moreover, the slope of process S depends on the degree of physical activity, with more sedentary days resulting in a slower build-up of sleep propensity and therefore a flatter slope. Process C is in turn controlled by an independent circadian (approximately 24 hours) oscillator and determines the timing of high and low sleep propensity throughout the day. The synchronization of the circadian rhythm depends on external cues (i.e., zeitgebers), with light being the primary stimulus. Other cues affecting process C are nutrition intake, temperature and exercise. Shift-work and jetlag are conditions where a disrupted circadian rhythm can interfere with sleep.
Appendix 3. 3P-model adapted from Spielman and Glovinsky 1991

The figure shows the 3P-model which is a theoretical framework of the factors causing and maintaining insomnia. It proposes that the presence of predisposing factors (i.e., personal, biological and social factors) may increase an individual’s vulnerability to developing insomnia. In addition, precipitating factors (e.g., disease, childbirth) are triggers causing the onset of insomnia which is represented in the graph by insomnia severity exceeding the threshold of clinical insomnia. Perpetuating factors (e.g., inappropriate cognitions, attitudes and behaviors) then ensure that, even after the provoking factor is reduced or eliminated, insomnia persists and becomes chronic.
Appendix 4. Micro-analytical model of insomnia adapted from Morin et al. 1993

The diagram shown consists of 4 components of insomnia (arousal, dysfunctional cognitions, maladaptive habits and consequences) that (reciprocally) influence each other. Arousal is frequently the cause of poor sleep and may occur as an emotional (e.g., anxiety), mental (e.g., concerns) or physical (e.g., pain) trigger. After several nights of disrupted sleep, people develop certain concerns and habits to cope with the sleep problems. These perceptions and behaviors may help reduce the effects of insomnia in the short term, but in the long term they appear to be incompatible with sleep and only exacerbate the problem resulting in negative consequences.
Combining cognitive behavioral therapy for insomnia and chronic spinal pain within physical therapy: a practical guide for the implementation of an integrated approach

ABSTRACT

The majority of people suffering from nonspecific chronic spinal pain (nCSP) report comorbid insomnia. However, in current treatment strategies for nCSP, insomnia is usually not addressed. Considering the bidirectional interaction between pain and sleep and its underlying psychophysiological mechanisms, insomnia may increase the risk of developing adverse physical and psychological health outcomes and should thus no longer be left untreated. As suggested by previous pilot studies, adding cognitive behavioral therapy for insomnia (CBT-I) to the contemporary evidence-based biopsychosocial physical therapy approach, may also improve pain outcomes in nCSP. This manuscript aims at providing practical guidelines on how hybrid physical therapy, including the combination of following components: (1) pain neuroscience education (i.e., to reconceptualize pain) and cognition-targeted exercise therapy (i.e., graded exposure to functional daily life movements), and (2) CBT-I (i.e., sleep psychoeducation, behavioral and cognitive therapy, correcting sleep hygiene, and relaxation therapy) can be deployed for the management of patients suffering from chronic spinal pain.

Impact statement:

Because of the major impact sleep disturbances have on pain and disability, insomnia as a comorbidity should no longer be ignored when treating patients with chronic spinal pain.

Manuscript word count: 5348
Chronic idiopathic low back or neck pain, failed back surgery syndrome (i.e. no resolution of symptoms despite anatomically successful surgery more than three years ago) and chronic whiplash-associated disorders present for at least 3 months, are referred to as nonspecific chronic spinal pain (nCSP). This disorder is highly prevalent and disabling, with severe personal and socioeconomic implications. The underlying pathophysiology of nCSP points toward alterations in the central nervous system, making it a complex and multidimensional sensation involving also psychological factors. Accordingly, treatments targeting local structures appear to have little effect in patients with nCSP. In contrast, contemporary cognitive-behavioral therapies (CBT) for nCSP, consisting of pain neuroscience education and cognition-targeted exercise therapy (CTET), among others, yields promising results. However, potentially associated comorbidities such as chronic insomnia generally remain untreated in physical therapy practice. Ignoring sleep complaints, though, might delay or impede effective pain management in a subgroup of nCSP patients with comorbid insomnia given that disturbed sleep increases pain sensitivity. Moreover, a good night’s sleep seems to increase daytime physical activity which may contribute to tackling nCSP. These factors emphasize the value of addressing sleep within therapy.

Chronic insomnia disorder is defined as unsatisfactory sleep, often associated with difficulties initiating and/or maintaining sleep in the presence of adequate opportunity and circumstances to sleep, and with consequences on daytime functioning. Additionally, sleep problems must be present more than 3 days a week for at least 3 months. Percentages reflecting the prevalence of clinical insomnia as a comorbidity in chronic pain conditions range from 53% to 90%. Also in persons reporting spinal pain, sleep problems are dominantly present with reported rates of 50% and more. In practice, therapy for chronic insomnia is often limited to pharmacological interventions and/or sleep education. However, in
addition to various adverse effects, evidence for the effectiveness of sleep medication appears limited, especially in the long-term.\textsuperscript{17,23} Furthermore, sleep hygiene as a stand-alone therapy, containing advice on behavioral and environmental factors, yields unconvincing results as well.\textsuperscript{27,28} The recommended first-line therapy for chronic insomnia is therefore cognitive-behavior therapy for insomnia (CBT-I).\textsuperscript{26,29} Indeed, this multicomponent treatment approach, encompassing sleep psychoeducation, time-in-bed restriction, cognitive and behavioral therapy (stimulus control) and relaxation training, appears to have manageable adverse effects and has been found to be effective in the long-term treatment of chronic insomnia.\textsuperscript{26,29}

\section*{Application of CBT-I in People with Chronic Pain and Comorbid Insomnia}

Various randomized controlled trials (RCTs) established the effectiveness of non-pharmacological sleep interventions (e.g., sleep hygiene, CBT-I) to enhance sleep outcomes in various chronic pain populations, but fail to demonstrate a consistent impact on pain.\textsuperscript{30} Moreover, the current lack of a holistic approach may increase the reliance to polypharmacy with potential risks of side effects and an amplification of the comorbidity.\textsuperscript{31} Therefore, the integration of a hybrid therapy, targeting both insomnia and chronic pain, seems appropriate to optimize nCSP management. Indeed, such an integrated approach, aiming at simultaneously addressing the physiological, psychological, and behavioral aspects of the coexisting complaints, could break the vicious cycle as improvement in one disorder might facilitate progress in the other. A long-term view in interpreting treatment effects is crucial in this context, given the essential but sometimes time-consuming role of cognitive and behavioral changes as of function of lifestyle factors.\textsuperscript{32}
Rationale behind a Hybrid Therapy Approach

Application of a Hybrid Therapy Approach in People with Chronic Pain and Comorbid Insomnia

Two pilot studies in a chronic pain population demonstrated the effectiveness of a therapy approach that combined CBT for sleep and pain on both sleep and pain-related parameters compared to waitlist controls.\textsuperscript{33,34} Additionally, findings from one RCT in elderly patients suffering from osteoarthritis supports the hypothesis that sleep management may beneficially impact pain and quality of life.\textsuperscript{35,36} Together, these findings provide a robust proof of concept to integrate a blended treatment approach in the management of patients with nCSP and comorbid insomnia, combining CBT-I and PNE with more active treatment modalities.\textsuperscript{37}

Bidirectional relationship between sleep and pain

While experimental and clinical studies demonstrate a bidirectional relation between central sensitization and sleep disturbances, accumulating evidence suggests that disturbed sleep is a more reliable predictor for pain development than the other way around.\textsuperscript{38,39} Consequently, in addition to being a perpetuating and intensifying factor for pain, insomnia also appears to be an etiological determinant.\textsuperscript{12} Thus, a vicious cycle is created in which sleep problems enhance pain sensitivity, leading to generalized hyperalgesia, which further results in an increased incidence or severity of sleep disturbances.\textsuperscript{33,40} Typically, however, sleep impairments and chronic pain are considered symptoms and consequently treated independently, disregarding possible common underlying interacting mechanisms.

Potential underlying mechanisms

The vital yet extremely complex homeostatic processes of both sleep and central sensitization implicate the involvement of many intricate, dynamic and multifaceted interactions.
Numerous mechanisms have already been suggested to explain the association between sleep and chronic pain however, the exact underlying mechanisms of this interaction have not yet been elucidated.\textsuperscript{12,13,41,42}

The balance of the immune system and the interrelated hypothalamus-pituitary-adrenal (HPA) axis is thought to be affected by disturbed sleep, shifting to a potentially glia-mediated pro-inflammatory state associated with stress and anxiety.\textsuperscript{12,13} This implies increased levels of pronociceptive cytokines, prostaglandins and nitric oxide contributing to a heightened pain sensitivity, characteristic of individuals with chronic pain.\textsuperscript{43} Also, increased adenosinergic activity at A\textsubscript{2A} receptors is suggested to affect nociceptive processing leading to hyperalgesia.\textsuperscript{13}

Furthermore, several monoaminergic systems appear to be involved in the altered pain modulation associated with sleep disturbance.\textsuperscript{13,41} Thus, a potential role is suggested for dopamine, a wake-promoting mediator, key neurotransmitter of the reward system and involved in endogenous analgesia. Increased pain sensitivity due to sleep deprivation may thereby be associated with lowered dopaminergic activity.\textsuperscript{13,41} Also, dysfunctional serotoninergic pathways, involved in both the sleep-wake system and endogenous pain modulation, could mediate the hyperalgesic effects following disrupted sleep.\textsuperscript{12,13} The orexinergic system and its related neuropeptides (orexin A and B) are involved in both the sleep-wake process and nociceptive transmission and modulation. It is therefore hypothesized that alterations in this system have a role in hyperalgesia resulting from poor sleep.\textsuperscript{13}

Furthermore, a decrease in available opioid receptors, which play a principal role in antinociceptive central pain modulation, as a consequence of sleep disturbances is suggested resulting in less responsivity to opioids and thus reduced pain inhibition, thereby facilitating pain in chronic diseases.\textsuperscript{12,13}
The neurohormone melatonin not only has a sleep-promoting effect controlled by light exposure, but it also has an analgesic and anti-inflammatory function, potentially providing a role in the relationship between sleep and pain.

In general, disrupted sleep causes increased sensitivity and responses to nociceptive stimuli, and thus exacerbates existing pain hypersensitivity, reducing treatment effectiveness and delaying recovery. Despite the fact that the results of large RCTs are still pending, implementing sleep management in physical therapy should be at the top of the research agenda given the major impact of sleep disorders on chronic pain and the high degree of comorbidity of these disorders. The purpose of this paper, therefore, is to provide practical guidance on the application of current evidence-based knowledge on the association between sleeplessness and chronic spinal pain.

Hybrid Therapy Approach in People with nCSP and Comorbid Insomnia

Before applying the hybrid CBT approach, a thorough screening of the patient should be completed in order to exclude underlying sleep (e.g., obstructive sleep apnea, restless legs syndrome) or psychological (e.g., depression) pathologies. Self-reported questionnaires (e.g., Insomnia Severity Index, Pittsburgh Sleep Quality Index, Epworth Sleepiness Scale, Brugmann Fatigue Scale) can be used, and if indicated, referral for a polysomnography should be considered. Screening techniques and the content of the anamnesis to determine the presence of insomnia are beyond the scope of this paper but have been previously described.

Communication is a crucial cornerstone throughout therapy, with the physical therapist implementing techniques of motivational interviewing aimed at developing and engaging the...
patient’s autonomy with respect to behavioral change.\textsuperscript{46} This process of change is linked to
the transtheoretical model of change in which six phases are dynamically passed through (i.e.,
precontemplation, contemplation, action, maintenance, relapse and termination).\textsuperscript{47} Further
underlying theoretical frameworks consists of various concepts such as the social cognitive
theory\textsuperscript{48}, self-determination theory\textsuperscript{49} and self-regulatory model\textsuperscript{50}, all of which share common
constructs. As such, the expectations of the patient about the implications of behavior change,
the patient’s perception of his or her self-efficacy (i.e., patient’s belief in his or her own ability
to change behavior) and the social context of the behavior are all addressed in these
theories.\textsuperscript{51} A Socratic communication method (i.e., open questions, empathic and reflective
listening) is applied to assess treatment acceptance, strengthen the patient-therapist
relationship and resolve ambivalence, allowing the individual’s intrinsic motivation to be
identified and reinforced.\textsuperscript{46,52} This way, therapeutic alliance and adherence are facilitated,
with the patient adopting an active role during therapy.\textsuperscript{53–56} However, in contrast to finding
positive outcomes in patients with low back pain resulting from a strong therapeutic alliance,
evidence that improved adherence in the short-term also affects therapy outcomes is lacking
to date.\textsuperscript{53,57}

An example of the practical implementation of the hybrid therapy approach applied to a case
can be found in the supplementary material.

\textbf{1. General Education}

\textbf{Pain Neuroscience Education}
Pain neuroscience education (PNE) entails the reconceptualization of pain to alter patients’ dysfunctional pain cognitions and disease perceptions in order to enable the application of active exercise therapy in a further therapy stage.\textsuperscript{58–60}

In an initial educational session, the purpose and course of acute pain in the nervous system as well as the changes that occur in chronic pain, including the mechanisms involved in central sensitization, are discussed based on a visual presentation (e.g., http://www.paininmotion.be/education/tools-for-clinical-practice).\textsuperscript{58,60} The content of the presentation intends to change the individual’s perception that pain is related and proportional to tissue damage. Through the acquired knowledge, pain should be disconnected from tissue damage by associating it primarily with altered processes in the brain, making it less threatening.\textsuperscript{61} After this educational session, patients receive a booklet and/or online module containing the taught material (e.g., http://www.paininmotion.be/education/tools-for-clinical-practice). Patients are asked to review this information at home to promote in-depth learning. In a subsequent session, questions are asked regarding the individual’s experiences and perceptions of pain (e.g., by using the self-reported Pain Catastrophizing Scale) to assess the patient’s is preparedness to move to a next therapy stage.\textsuperscript{62} The subject’s responses are then discussed and misinterpretations are corrected to ensure that the information received about pain is optimally implemented during CTET.

The effectiveness of PNE in improving both physical and psychological factors in various chronic pain conditions has been described previously.\textsuperscript{60,63} The combination with a physical intervention (e.g., exercise therapy) is recommended to optimize the synergetic treatment outcome.\textsuperscript{7,60,64,65}

\textbf{Sleep Psychoeducation}
The main objectives of sleep psychoeducation are to inform, correct patients’ dysfunctional beliefs and obtain realistic expectations regarding sleep and its relationship to pain so that therapy components can be applied and therapy adherence is encouraged. Thus, it provides patients with a solid and science-based understanding of natural sleep and the underlying psychological and behavioral factors in the development of chronic insomnia. In one session, basic concepts of normal sleep and its corresponding sleep mechanisms are explained using an illustrated presentation. Appendices 1 to 4 detail the sleep principles essential within sleep psychoeducation.

2. Behavioral Therapy

Time-in-Bed Restriction Therapy

Sleep Diary

To obtain a subjective estimate of an individual’s sleep duration, waking periods during the night, and the timing of these parameters relative to a 24-hour period, a sleep diary is utilized during an initial observation phase. The patient is instructed to complete the sleep diary during an initial week without making any changes to the actual sleep pattern, other than avoiding clock-watching, to obtain an accurate baseline for applying further therapy. Indeed, a sleep diary is a valuable tool for a physical therapist to evaluate self-reported insomnia based on sleep efficiency (SE; proportion of time in bed spent asleep), sleep quality, feelings upon waking and throughout the day. Additional information on physical activity and the intake of medication, alcohol, nicotine, and caffeine could also be noted in the diary, as these are important factors affecting sleep (see also sleep hygiene). The patient is additionally asked to indicate the time points when fatigue or sleepiness occurred. To be complete, information
about experiencing pain may be recorded in the sleep diary. To obtain complete information on relevant sleep aspects, discussion of the sleep diary with the patient is appropriate to further nuance the sleep and wake experiences.

A panel of experts has developed a standardized sleep diary (Consensus Sleep Diary, CSD, https://consensussleepdiary.com/) of which the psychometric properties support validity, clinical utility and usability.\textsuperscript{66,67} Alternative formats of sleep diaries are used as well, including time charts, clock faces, or electronic diaries. Figure 1 shows an example of a blank sleep diary based on a time chart that can be used in practice. Instructions for completing this sleep diary can be found in Box 1.

**Interpretation of the Score Form**

The sleep diary is scored together with the patient during a subsequent session by calculating the average values of the various sleep parameters using a score form. Figure 2 shows an example of a blank score form and Box 2 provides the instructions for calculating the various sleep parameters. Both the patient and physical therapist thus gains insight into the patient's subjective sleep pattern, providing a broader perspective and subsequently specific behavioral changes can be targeted. The sleep diary and its accompanying score form are therefore used as data source on a week-to-week basis throughout further therapy for two reasons: (1) to monitor time-in-bed restriction, and (2) to observe treatment-related changes in sleep outcome parameters.

**Time-in-bed restriction**

Time-in-bed restriction aims to reduce time spent awake in bed, achieve a state of mild sleep deprivation by initially restricting the time spent in bed to the average total sleep time, thus increasing the homeostatic sleep pressure, and realigning the circadian rhythm to this process.
This objective is reached by altering perpetuating coping strategies, thereby improving sleep latency and general sleep quality.\textsuperscript{68}

By scoring the sleep diary, SE is calculated which represents the possible mismatch between time spent in bed and total sleep time, providing the primary indicator for the application of time-in-bed restriction. Based on the SE percentage, an appropriate sleep window (i.e., target amount of sleep opportunity in bed) is defined according to the average total sleep time for the past week. First, the hour of awakening is determined and then a target time for going to bed is obtained by backward counting the number of hours of effective sleep from the time of awakening in the morning (e.g., a sleep window of 7 hours and 30 minutes could result in a total time in bed from 11.00 pm to 6.30 am).

The following instructions are proposed concerning SE: (1) if SE is < 85\%, a new sleep window is determined by making the total time in bed equal to the mean total sleep time of the past week (sleep window \( \geq 5 \) hours because of safety and health concerns), (2) if SE is 85 - 90\%, the sleep window is retained; and (3) if SE is > 90\%, the sleep window is extended with 15 minutes, allowing for a potential increase of total sleep time. So, according to these guidelines, a time-in-bed restriction is obtained if SE is < 85\%.\textsuperscript{69}

As time-in-bed restriction can result in mild sleep deprivation, and consequently daytime sleepiness, it is important to explain the purposes of sleep-wake scheduling (reducing the amount of awake time spent in bed rather than shortening sleep duration; increasing sleep propensity) to the subject. In addition, the physical therapist should evaluate the individual’s intrinsic motivation by Socratic communication and establish a trusting therapeutic relation before implementing time-in-bed restriction in therapy.\textsuperscript{66} As a consequence of sleep deprivation, daytime sleepiness may be provoked, which can indicate the therapy
effectiveness as it reflects increased sleep propensity. However, the enhanced sleepiness seem to occur in the first weeks after the introduction of the time in bed restriction and usually returns to a baseline level thereafter.  

During further therapy, each session starts with reviewing the sleep diary, interpreting SE and repeating the procedure of time-in-bed restriction by altering the sleep window if necessary. Indeed, as SE increases over time, the sleep window is gradually extended, allowing an increase in total sleep time until an optimal equilibrium is found between sleep duration and efficiency, with possible beneficial effect on pain parameters.  

Sleep compression

Sleep compression is a milder alternative to time-in-bed restriction since sleep opportunity is gradually reduced over several weeks rather than radically curtailing sleep early in therapy. 69,71 Compression of time in bed is attained by either advancing wake-up time or delaying bedtime until the average baseline sleep time is reached. This strategy may be applied in anxious patients and individuals in whom time-in-bed restriction is not tolerated because of the abrupt decrease in sleep opportunity. However, sleep compression also entails some disadvantages as it may take longer for the insomnia symptoms to subside, possibly resulting in frustration and/or a lack of therapy adherence. More specifically for nCSP patients, this approach may be appropriate to prevent an increase in pain sensitivity given the hyperalgesic response caused by sleep deprivation. 71

Medication

Analgesics
Sleep disturbance negatively affect the efficacy of pain medications (e.g., opioids), increasing the risk of dose escalation and consequently dependence. In patients with chronic pain, it is recommended that the dosage and timing of intake of analgesics should be determined and adjusted in consultation with a physician to minimizing its sleep-disrupting effects.

Sedative Hypnotics

Tapering and subsequently discontinuing the intake of sleep medications are among the therapy objectives. Indeed, the risk of becoming dependent on hypnotics increases with frequent and prolonged use. Before starting to withdraw the medication, the therapist should check whether the patient is motivated to stop the intake of medication. Since there are risks for adverse effects such as rebound insomnia, if sleep medication intake is stopped suddenly and without medical supervision, it should be phased out gradually. For this reason, it is necessary to collaborate with the prescribing physician to determine if and how sleep medication can be phased out. If the patient takes a combination of different sleep agents, the physician decides which medication will be discontinued first. A gradual tapering schedule is than created in consultation with both the physician and the patient, consisting of a 25% reduction in medication, at intervals of 1 to 2 weeks, until the lowest available dose is reached.

Stimulus Control

Stimulus control includes behavioral instructions, based on classical and operant conditioning that counteract inconsistent pain and sleep behavior.

Sleep
Patients with chronic insomnia often develop a classically conditioned response, namely between the bed(room) and lying awake. Therefore, stimulus control regarding sleep aims to restore the positive association between the bed(room) and sleep and to (re)establish a regular sleep-wake pattern.\(^{75-77}\) Specific sleep related stimulus control instructions to the patients are the following: (1) only go to bed if you feel sleepy, (2) get out of bed when unable to sleep within 15 minutes, (3) use your bed only for sleeping and sexual activities, (4) avoid naps during the day, and (5) maintain a consistent sleep-wake schedule.

**Pain**

Specific to nCSP, inactivity and inappropriate protective or safety behaviors (e.g., adjusting or avoiding bending forward because it provoked pain in the past; co-contracting stabilizing muscles to protect the spine) may be applied as coping strategies and need to be targeted during exercise therapy (see CTET).\(^{59,73}\)

In addition, pain and its associated reactions may indirectly contribute to sleep issues. Nighttime physical discomfort, such as spinal pain, can interfere with sleep leading to a loss of control, pain catastrophizing and consequently pre-sleep cognitive hypervigilance. Extending time in bed or resting in bed during the day as a function of pain or fatigue rather than effective sleepiness, may also reduce SE and relegate the bed(room) to a weaker cue for sleep in patients with CSP. Addressing these issues during therapy is indicated.

### 3. Cognitive Therapy

The aim of cognitive therapy is to change an individual’s dysfunctional beliefs and expectations about sleep and/or pain by verbal interventions and behavioral experiments. Modifying these perceptions reduces emotional and somatic arousal, addresses barriers for the required
behavioral change, and may subsequently improve sleep and/or pain parameters, thus breaking the vicious cycle between arousal and sleep problems and/or pain. The therapist guides patients to reevaluate their thinking by providing key messages (e.g., ‘insomnia is not the (only) cause of all daytime dysfunctions, there may be other explanations for these deficiencies’) and behavioral experiments in the form of homework (e.g., testing out the effect of active versus passive behavior as a function of pain). Thus, a more realistic and rational view of sleep and/or pain is achieved. In addition, problem-solving training, such as noting problems and the associated first step of the solution before bedtime, and acceptance-based therapies can reduce arousal.

General education (PNE and sleep psychoeducation) constitutes an essential part of the cognitive component within therapy and aims to facilitate the implementation of more active interventions driven by patient self-efficacy. During the initial therapy phase, education is provided that serves as the foundation for the subsequent treatment progression, turning it into a continuous process that is referred to repeatedly. In the context of CTET, pain education ensures the ability to apply a time-contingent rather than a pain-contingent approach. This involves performing an exercise to the frequency and intensity agreed upon during therapy, without modifying or stopping when experiencing pain.

Insomnia and spinal pain are both more prone to evolve into chronic diseases when an individual perceives this complaints as a loss of control and is concerned about the consequences. Indeed, dysfunctional cognitive responses feed the vicious cycle of insomnia and pain. Increased emotional stress produces a state of both emotional and physiological hypervigilance, possibly enhanced by (presleep) somatic arousal caused by pain,
which all adversely affects sleep. An essential objective of cognitive therapy is therefore to enhance the feeling of control and to strengthen coping skills when dealing with sleep problems and their consequences.

**Sleep Hygiene**

Sleep hygiene encompasses a range of advice related to lifestyle (e.g., physical activity, diet, substance use) and environmental factors (e.g., temperature, noise, light) that, although often not the primary cause of chronic insomnia, may contribute to the perpetuation of sleep problems. Educating the patient about the relative risks of these factors can help improve treatment progress. Thus, the purpose of sleep hygiene instructions is to tackle unfavorable sleep behaviors by providing appropriate information to improve sleep quality. Box 3 shows the advices provided to the patient regarding sleep hygiene. Some of these factors overlap with the components of time-in-bed restriction and stimulus control.

Caffeine consumption and smoking of nicotine are detrimental to sleep since both stimulate the central nervous system. Alcohol, in turn, is a suppressor of the central nervous system resulting in a short sleep onset latency, but reduced sleep duration and poorer sleep quality due to withdrawal symptoms. Hence, the use of these substances before bedtime is discouraged. Additionally, intensive exercise within an hour before bedtime is not recommended because it activates the autonomic nervous system and increases body temperature, leading to a delay in the onset of sleep. However, energy expenditure from physical activity does promote sleep when exercise is performed at other times of the day and is also beneficial in reducing stress.

**4. Relaxation training**
(Hyper)arousal is known as one of the factors that contributes to the onset and maintenance of insomnia. In turn, poor sleep can reduce a person's ability to cope with everyday stressors. Relaxation therapy therefore aims to reduce somatic and/or cognitive arousal and its associated autonomic stress response in function of indirectly improving both sleep quality and pain. Practicing relaxation techniques before bedtime is encouraged in the presence of pre-sleep arousal, where the deactivation of the stress system through relaxation can be further complemented by adapting evening activities and implementing breaks (i.e., mental or physical rest) throughout the day. Learning a relaxation technique is a time-consuming, progressive process that requires daily practicing for a period of a few weeks. The selection of the applied relaxation technique depends on the presence of cognitive or somatic arousal. In order to reduce racing thoughts (i.e., cognitive arousal), attention-oriented techniques as visualization or thought stopping techniques are appropriate to focus on more pleasant images or thoughts. In the case of dominant muscle tension, progressive muscle relaxation is indicated, whereby certain muscle groups are alternately contracted and relaxed. Control of physiological parameters through auditory or visual feedback, referred to as biofeedback, is another therapy that can be recommended in somatic arousal. The choice of relaxation technique is further determined by the patient's preference. Previous positive or negative experiences with relaxation may additionally influence patient's perspectives, so questioning and considering these factors is critical. If stress has a profound impact on the individual, more comprehensive stress management offers a valuable complement to relaxation therapy. More detailed information on relaxation and stress management can be found elsewhere.

5. Cognition-targeted exercise therapy
Once a person suffering from CSP has adopted the biopsychosocial principles of the PNE, CTET can be initiated aiming to alter the patient’s pain memory in the brain. The amygdala as part of the pain neuromatrix is a key brain area involved in the memory of painful movements. The overactivation of the pain neuromatrix due to neuroplasticity, results to maladaptive changes in response to stimuli. By cooperating with other brain regions such as the anterior cingulate cortex and hippocampus, pain memory directs protective safety behaviors in response to movements previously experienced as painful. As such, this long-term pain memory can interpret certain daily life movements as threatening, causing pain and resulting in an inappropriate safety response (e.g., antalgic postures, co-contraction of stabilizing muscles) that interferes with a natural and functional way of moving. Cognition-targeted exercise therapy therefore aims to systematically desensitize the central nervous system by substituting the dysfunctional pain memory through enhancing pain-coping strategies via repeated and progressive exposure to certain challenging movements or postures. Again, appropriate communication forms a crucial therapy component whereby the cognitions, perceptions and expectations of the patient regarding a specific movement and the possible associated pain are constantly questioned. So, based on the patient’s individual fear of movement, a time-contingent, individual approach is applied whereby the degree of challenge of the exercises is built up progressively, starting from simple analytical to more dynamic and functional movements. The patient is also encouraged to do daily home exercises to further alter the maladaptive pain memory. Examples of cognition-targeted exercises and concurrent communication were described elsewhere. Therapeutic effects of exercise training appear to be manifested centrally, including psychological, neurophysiological and cognitive modifications, rather than being directly attributed to the rectification of a biomedical deficiency of the musculoskeletal system.
Thus, the outcome of motor control training in both neck and back pain appears not to be superior compared to other types of exercise. Instead, motor control training might be counterproductive by encouraging improper safety behavior, increasing fear of movement, and consequently pain when performing a movement without co-contraction of the stabilizing muscles.

6. Relapse prevention

Relapse prevention seeks to avoid long-term recurrence of symptoms by reiterating the therapy principles and identifying potential risk factors or situations. It thus assumes the facilitation of the further development or maintenance of the behavioral change brought about as a function of more appropriate coping strategies. If necessary, the behavior change theories can be invoked again in order to prevent further relapse. The therapist evaluates whether the patient’s objectives regarding sleep and pain, set in the first session, are achieved, and if not, what could be the underlying causes. Furthermore, the patient is advised to practice the acquired competencies to further optimize pain parameters and sleep (and wake) quality.

With respect to chronic spinal pain, relevant home exercises and possible paths for further progression are discussed. The emphasis during the sleep evaluation is on the progress in general without focusing on a bad night or week. Thus, more than achieving good sleep parameters and scores, sleep therapy aims to improve patient’s sleep perception by accepting that sleep (and wake) may not be perfect on a daily basis, resulting in less concern and anxiety about sleep. Booster sessions (e.g., in person, via telephone) may be appropriate to prevent relapse.
How to put Hybrid Therapy into Practice?

Typically, CBT-I is delivered by psychologists or health providers with training in sleep medicine and behavioral and/or cognitive therapy. However, to date there is a lack of specialists who can accurately treat the vast number of patients with insomnia. Considering up to 90% of patients with chronic pain exhibit impaired sleep, providing CBT-I within the physical therapy practice may be a beneficial addition to compensate for this deficit in sleep specialists. Although many physical therapists consider sleep to be an important factor likely to affect therapy outcomes, a lack of knowledge on this topic seems to account for the fact that sleep is not yet adequately addressed in daily physical therapy practice. Integrating information on sleep into the entry-level curriculum of physical therapy students and providing continuing training opportunities to practicing physical therapists appears to be a crucial step in optimizing treatment regarding insomnia. As one could argue the implementation of CBT-I in physical therapy, a recent study has established that physical therapists provided with specific training (e.g., on behavior change theories) are able to effectively carry out cognitive behavioral therapy in clinical practice. In the future, the introduction of a certificate of competence for CBT-I specialists, as an extension of a clinician’s practice license, may be a useful option for ensuring the quality of therapy. However, proper billing and reimbursement of CBT-I still appears to pose problems for physical therapists. In contrast, clinicians practicing in the mental health field, providing cognitive and/or behavioral treatments, are familiar with and accustomed to billing that is consistent with mental health rates and co-payments. As the implementation of CBT-I in the health care systems seems to vary across the world, a need exists for more accessible, multidisciplinary incorporation of CBT-I within all health care systems.
Based on the dominant complaint and the patient’s history, the focus within therapy may be on sleep or pain. However, the concept of simultaneously addressing sleep and pain, by alternating exercise with sleep therapy sessions, remains the starting point of the approach. Furthermore, the practical implementation of the various therapy components should be tailored to the patient and the possibilities within the therapeutic setting. Thus, the format of the sessions may vary for cost-effectiveness, providing individual, group sessions, or a combination of both. Additionally, group sessions promote interaction compared to an individual approach. The option of online sessions rather than live sessions may also offer a solution regarding cost-effectiveness. Dose and duration of therapy sessions vary across literature. Previous findings indicated that 4 or more individual CBT-I sessions are more effective in treating insomnia compared to fewer sessions or self-help interventions. The duration of a session may range between 30 minutes and 2 hours. Table 1 presents a proposal of an alternating therapy structure between sleep- and pain-focused sessions spread over 14 weeks. Figure 3 shows an example of the substantive organization of the various CBT-I components over 6 sessions.

Contra-indications for the application of CBT-I

A number of contra-indications are kept in mind to use CBT-I within nCSP management. In the presence of a sleep pathology, a severe addiction or mental disorder that has not yet been treated, the absence of negative conditioning about sleep, and insufficient motivation for behavioral change are considered contraindications for applying CBT-I. Adequate screening and referral if required are indicated to ensure optimal therapy for the patient. Yet, providing
sleep education and hygiene in this population remains recommended even in case of these contra-indications.\textsuperscript{100}

Conclusion

Growing evidence for the association between sleep and pain indicates that this comorbidity should no longer be ignored in the treatment of patients with chronic spinal pain. A therapy approach in which both chronic spinal pain and sleep are addressed seems appropriate. To meet this challenge, the current evidence-based contemporary neuroscience approach consisting of PNE and CTET is combined with CBT-I in order to target pain, disability and sleep complaints.

Author Contributions

Concept/design: (blinded)

Drafting/revising article: (blinded)

Final approval before submission: (blinded)

Role of the Funding Source

This paper was funded by the (blinded). The funder played no role in the design, conduct, or reporting of this study.
Conflict of interest

(Blinded) is involved in the training of CBT-I for psychologists at (blinded), by (blinded).

(Blinded) authored a (blinded) book on (blinded).
References


8. Moseley GL. Joining Forces – Combining Cognition-Targeted Motor Control Training with Group or Individual Pain Physiology Education: A Successful Treatment For


549 17. Riemann D, Baglioni C, Bassetti C, et al. European guideline for the diagnosis and


34. Tang NKY, Goodchild CE, Salkovskis PM. Hybrid cognitive-behaviour therapy for individuals with insomnia and chronic pain: A pilot randomised controlled trial. *Behav*


700  77. Morgenthaler T, Kramer M, Alessi C, et al. Practice Parameters for the Psychological


Table 1. Practical organization of therapy sessions over a treatment period of 14 weeks

<table>
<thead>
<tr>
<th>WEEK</th>
<th>THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2</td>
<td>PNE</td>
</tr>
<tr>
<td>3</td>
<td>CBT-I</td>
</tr>
<tr>
<td>4</td>
<td>CBT-I</td>
</tr>
<tr>
<td>5</td>
<td>CBT-I</td>
</tr>
<tr>
<td>6</td>
<td>CBT-I</td>
</tr>
<tr>
<td>7</td>
<td>CTET</td>
</tr>
<tr>
<td>8</td>
<td>CBT-I</td>
</tr>
<tr>
<td>9</td>
<td>CTET</td>
</tr>
<tr>
<td>10</td>
<td>CBT-I</td>
</tr>
<tr>
<td>11-14</td>
<td>CTET</td>
</tr>
</tbody>
</table>

PNE, pain neuroscience education (group session + online session + individual session); CTET, cognition targeted exercise therapy; CBT-I, cognitive behavioral therapy for insomnia.
**Figure 1. Sleep diary**

![Sleep Diary Diagram]

**Figure 2. Score form**

![Score Form Diagram]

<table>
<thead>
<tr>
<th>Time of going to bed</th>
<th>Sleep / nap</th>
<th>Awake out of bed</th>
<th>Awake in bed</th>
<th>Alcohol</th>
<th>Physical activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time of getting up</td>
<td>Sleepiness</td>
<td>Fatigue</td>
<td>Nicotine</td>
<td>Medication</td>
<td>Caffeine</td>
</tr>
</tbody>
</table>
Figure 3. Timeline for applying cognitive behavioral therapy for insomnia

<table>
<thead>
<tr>
<th>Week from Monday - Tuesday</th>
<th>Tuesday - Wednesday</th>
<th>Wednesday - Thursday</th>
<th>Thursday - Friday</th>
<th>Friday, Saturday</th>
<th>Saturday - Sunday</th>
<th>Sunday - Monday</th>
<th>MEAN of the week</th>
<th>8 hours (mean/SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time in bed (TIB)</td>
<td>sleep education</td>
<td>behavior therapy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep onset latency (SOL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wake after sleep onset (WASO)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total wake time (TWT = SOL + WASO)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total sleep time (TST = TIB-TWT)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep efficiency (%) (TST/7 x 100)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep quality (/30)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daytime feeling (/30)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Main components per session:

- Session 1 - sleep psychoeducation + explanation on how to fill in the sleep diary
- Session 2 - explanation on how to score the sleep diary and interpret the score form + time-in-bed restriction
- Session 6 - relapse prevention
Session 3 - reevaluation of sleep window + possible extension/reduction of sleep window + stimulus control

Session 4 - reevaluation of sleep window + possible extension/reduction of sleep window + sleep hygiene

Session 5 - reevaluation of sleep window + possible extension/reduction of sleep window + relaxation training

Session 6 - evaluation + relapse prevention

Box 1. Instructions on how to fill in a sleep diary

- Complete the items about the previous night in the sleep diary at a set time in the morning, about 20-30 minutes after waking to limit the influence of sleep inertia (i.e., the period of reduced alertness and/or performance after waking up).

- In the evening, enter in the sleep diary any notable additional items that occurred during the day (i.e., intake of alcohol, caffeine, napping, etc.).

- Put your sleep diary in a visible place to reduce the chance of forgetting to fill it out. If you do forget, leave the boxes for that night open rather than filling them in later.

- Do not check the clock during the night. The sleep diary is an estimate of your sleep and therefore subjective.
- Each hour is divided into 4 boxes, each representing 15 minutes. All times requested should be rounded according to these 15-minutes boxes.

- For the time you go to sleep and turn off the light, put a downward arrow (↓) on the dividing line between the 2 boxes for the corresponding hour.

- For the time you get up in the morning, put an upward arrow (↑) on the dividing line between the 2 boxes for the corresponding hour.

- If you read, work or watch television in bed during the evening, indicate this with a vertical line at the time you start doing so (followed by the downward arrow when you turn off the light and try to sleep).

- Color all boxes for the hours you slept (according to your feeling) at night or during the day (i.e., napping).

- Arce the boxes corresponding to the time in bed without sleeping (e.g., if you spent 15 minutes awake in bed, arce the corresponding box).

- Mark the intake of medication (M) caffeine (C), alcohol (A), or nicotine (N) and physical activity (P) in the corresponding boxes. Further specifications can be given under ‘treatment and remarks’ (e.g., name and dose of medication).

- Mark the times when you felt sleepy (S; subjective experience of a need for sleep and an effort to stay awake, resolved by sleep) or fatigued (F; feeling of exhaustion, often caused by muscular, emotional or mental exertion and elevated by rest) in the corresponding boxes.

- Sleep disturbing factors (e.g., pain, environmental noise) may be additionally noted under ‘treatment and remarks’. Other notable issues (e.g., intense pain, vacation) may also be listed under this heading.
- **Time in bed (TIB):** count the number of boxes of 15 minutes between going to bed (↓) and getting up (↑). Time out of bed during the night is not counted.

- **Sleep onset latency (SOL):** count the number of shaded boxes of 15 minutes between going to bed (↓) and the first colored box.

- **Wake after sleep onset (WASO):** count the number of shaded boxes between the first colored box and getting up in the morning (↑).

- **Total wake time (TWT):** sum up the SOL and WASO.

- **Total sleep time (TST):** subtract the TIB from the TWT.

- **Sleep efficiency (SE):** divide the TST by the TIB and multiply by 100 to obtain a

  Rate your sleep quality and rested feeling (awakening quality) from 0 to 10 (0 = not good, 10 = very good) at the time you complete the sleep diary.

  Rate your feelings during the past day from 0 to 10 (0 = not good, 10 = very good) in the evening.

---

**Box 2. Instructions on how to calculate sleep parameters of score form**
Box 3. Sleep Hygiene Instructions

- Avoid caffeine consumption 4 to 6 hours before bedtime.
- Do not smoke before going to bed or when you wake up at night.
- Avoid alcohol consumption 4 to 6 hours before bedtime.
- Avoid heavy meals before bedtime.
- Avoid intense training 2 hours before bedtime.
- Provide comfort to your bedroom.
- Avoid extreme temperatures in your bedroom.
- Keep your bedroom quiet and dark.
- Avoid active interacting with screens 1 hour before bedtime.

Percentage, i.e., \((\text{TST} / \text{TIB}) \times 100\).

- Calculate the average for the week of each sleep parameter by dividing the sum of every row by 7.
- Because each box represents 15 minutes, divide the number of boxes by 4 to obtain the number of hours. The number before the comma indicates the number of hours, the number after the comma indicates the number of quarters in decimal places (i.e. 0.25 = 15 minutes, 0.5 = 30 minutes and 0.75 = 45 minutes).
Appendix 1. Hypnogram

The stable sequence of the different sleep cycles (x-axis) and stages (y-axis) of a healthy night’s sleep are represented in a hypnogram, reflecting an individual’s personal sleep architecture. The time span of one cycle typically ranges from 90 to 120 minutes, resulting in a number of...
4 to 6 cycles per night. During one cycle, an individual passes through the various sleep stages. Thus, in a first non-rapid-eye movements (REM) phase (N1), the transition from wake to sleep occurs. It concerns a light sleep that lasts only a few minutes before progressing to the next stage, the second non-REM phase (N2). During this phase muscle tension will continue to decrease. In addition, heart rate and respiration also slow down in preparation for deep sleep (N3) in which a low frequency will eventually be reached. Finally, during REM sleep, heart rate and respiration become irregular. Furthermore, considerable brain activity occurs whereby information and experiences of the past day are ordered and processed. A shift in sleep phases during the night can be observed, i.e., a dominant slow wave sleep (N3) at the beginning of the night developing into more dominant REM sleep by morning. Moreover, this figure also shows that short awakenings throughout the night are a natural phenomenon and usually occur unconsciously due to amnesia during the night.

Appendix 2. Two-process model of sleep regulation adapted from Borbely 1982

The graph shows the two-process model of sleep regulation representing the timing of the sleep-wake alternation resulting from two interacting processes: a homeostatic process (process S) that reflects the sleep-dependent accumulation of sleep pressure, and an
endogenous sleep-independent circadian rhythm (process C) tracking the environmental time. Process S thus regulates the sleep depth and intensity characterized by a monotonous increase in sleep propensity during wake and a subsequent exponential decline during sleep.

Taking a daytime nap consequently reduces sleep pressure, which is likely to lead to problems initiating sleep around bedtime and lighter sleep during the night. Sleep deprivation, on the other hand, results in a buildup of sleep homeostasis, causing sleep debt. Sleep homeostasis can be optimized by going to bed only when sleep pressure is sufficiently present. As a function of this, time in bed restriction may be appropriate, as this will minimize waking time in bed and thus delay the time to sleep. Moreover, the slope of process S depends on the degree of physical activity, with more sedentary days resulting in a slower build-up of sleep propensity and therefore a flatter slope. Process C is in turn controlled by an independent circadian (approximately 24 hours) oscillator and determines the timing of high and low sleep propensity throughout the day. The synchronization of the circadian rhythm depends on external cues (i.e., zeitgebers), with light being the primary stimulus. Other cues affecting process C are nutrition intake, temperature and exercise. Shift-work and jetlag are conditions where a disrupted circadian rhythm can interfere with sleep.
Appendix 3. 3P-model adapted from Spielman and Glovinsky 1991

The figure shows the 3P-model which is a theoretical framework of the factors causing and maintaining insomnia. It proposes that the presence of predisposing factors (i.e., personal, biological and social factors) may increase an individual’s vulnerability to developing insomnia. In addition, precipitating factors (e.g., disease, childbirth) are triggers causing the onset of insomnia which is represented in the graph by insomnia severity exceeding the threshold of clinical insomnia. Perpetuating factors (e.g., inappropriate cognitions, attitudes and behaviors) then ensure that, even after the provoking factor is reduced or eliminated, insomnia persists and becomes chronic.
Appendix 4. Micro-analytical model of insomnia adapted from Morin et al. 1993

The diagram shown consists of 4 components of insomnia (arousal, dysfunctional cognitions, maladaptive habits and consequences) that (reciprocally) influence each other. Arousal is frequently the cause of poor sleep and may occur as an emotional (e.g., anxiety), mental (e.g., concerns) or physical (e.g., pain) trigger. After several nights of disrupted sleep, people develop certain concerns and habits to cope with the sleep problems. These perceptions and behaviors may help reduce the effects of insomnia in the short term, but in the long term they appear to be incompatible with sleep and only exacerbate the problem resulting in negative consequences.