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Abstract

Objective

Sleep disturbance in chronic pain is common, occurring in two thirds of patients. There is a complex relationship between chronic pain and sleep; pain can disrupt sleep and poor sleep can exaggerate pain intensity. Poor sleep and pain can also impact on both depressive symptoms and attention to pain. This study aims to evaluate the relationship between chronic pain and sleep, and the role of mood and attention.

Methods

Chronic pain patients, recruited from a secondary care outpatient clinic, completed self-report measures of pain, sleep, depressive symptoms and attention to pain. Hierarchical regression and structural equation modelling were used to explore the relationships between these measures. Participants (n = 221) were aged between 20 and 84 (mean = 52) years.

Results

The majority of participants were found to be ‘poor sleepers’ (86%) with increased pain severity, depressive symptoms and attention to pain. Both analytical approaches indicated that sleep disturbance and pain severity were not directly associated. Instead, the two parameters were associated via the indirect effects of mood and attention to pain.

Conclusions

The results indicate that sleep disturbance may contribute to clinical pain severity indirectly though changes in mood and attention. Prospective studies exploring lagged

associations between these constructs could have critical implications for the treatment of chronic pain.

Keywords: Sleep, Chronic Pain, Depression, Psychology, Attention

Exploring the associations shared by mood, pain-related attention and pain outcomes related to sleep disturbance in a chronic pain sample

Introduction

Sleep disturbance is reported by 65% of chronic pain patients (Deardorff, 2005), but its exact nature varies across different chronic pain populations, and can include difficulty with sleep initiation or maintenance, early awakenings, and fragmented or unrefreshing sleep (Cohen, Menefee, Doghramji, Anderson, & Frank, 2000; Perlis et al., 1997). Conventionally, pain and sleep have been thought to share a bi-directional and dynamic relationship; pain can disrupt sleep and poor sleep can exaggerate pain intensity. However, recent evidence suggests that this relationship might not be as straightforward as originally conceived (Tang, Goodchild, Sanborn, Howard, & Salkovskis, 2012). Evidence is starting to emerge proposing that sleep has a stronger temporal effect i.e. sleep disturbance influences pain, more than pain intensity influences sleep (Finan, Goodin, & Smith, 2013; Tang et al., 2012). Furthermore, it is becoming apparent that the relationship between sleep and pain may be mediated by a number of other factors including attention and mood.

Chronic pain is an endogenous source of stress, which can act to impair physiological, endocrinological and cognitive systems (Hart, Wade, & Martelli, 2003). The chronic overstimulation of the central nervous system may cause alterations in homeostatic wake-sleep cycles. Sleep disturbance may compromise the reparative functions associated with sleep, causing impaired healing and increased pain sensitivity (Lautenbacher, Kundermann, & Krieg, 2006). Furthermore, sleep disturbance is linked to maladaptive coping mechanisms as it can lead to both depression and anxiety, especially in chronic pain patients (Call-Schmidt & Richardson, 2003).

Environmental stimuli requiring an immediate behavioural response are typically prioritized and captured by our attentional focus (Norman & Shallice, 1986). In chronic pain patients, excessive attention to pain may play a part in the distress experienced. Attention to acute pain is often adaptive as it promotes escape from potentially dangerous situations. However, in chronic pain it can be difficult to disregard painful sensations. Constant interference can become maladaptive and the threat of pain can be difficult to disengage from (G. Crombez, Eccleston, Baeyens, & Eelen, 1998). This heightened focus on pain-related stimuli can lead to the development of attentional biases whereby patients become hyper-vigilant and prioritise the focus of their attention on pain stimuli. In states of hyper-vigilance patients may become more somatically-focused, monitoring their body for changes in pain sensation and symptoms. These biases predict how much pain will interfere with attentional resources, and can serve to maintain chronic pain states (Geert Crombez, Eccleston, Van den Broeck, Goubert, & Van Houdenhove, 2004).

Patients frequently seek pharmacological remedies, or behavioural adaptations to reduce the constant interference of pain in their daily lives. Often the long-term nature of chronic pain and its protracted treatment is difficult for the patient to understand and accept. As more avenues are explored and fail, both frustration and depressive symptoms may develop. Constant worry can interfere with both cognition and behaviour, causing patient's to ruminate over their self-evaluation and the threat of pain (Aldrich, Eccleston, & Crombez, 2000). As these 'pain-related fears' build, patients will begin to catastrophise (e.g., panic about future interactions with pain-related stimuli), further heightening attention to pain and promoting a vicious cycle (McCracken, 1997). Overall, attentional direction and focus are critical in modulating affective pain experiences, but how these factors are influenced by concomitant factors such as sleep and mood disturbance remains unclear.

Evidence now supports the notion that sleep and pain share a reciprocal relationship, the temporal direction of which might be stronger in sleep > pain direction. Sleep disturbance is seen to increase the likelihood of new incidences of chronic pain (Gupta et al., 2007; Odegard et al., 2011). Furthermore, for existing chronic pain cases, sleep disturbance is associated with maintaining symptoms and worsening both symptom severity and prognosis (Tang et al., 2012; Tang & Sanborn, 2014). Finan and colleagues (2013) suggested that sleep problems might not merely be a simple consequence of chronic pain, but perhaps directly alter the central pain processing mechanisms implicated in the pathophysiology of chronic pain. The role of attention and mood in this relationship remain poorly understood.

Chronic pain patients with co-morbid sleep difficulties report greater pain severity and tend to focus their attention more on pain. This heightened attention is also predictive of greater sleep disturbance (Affleck, Urrows, Tennen, Higgins, & Abeles, 1996). Similarly, negative mood has been found to mediate the association between sleep and pain severity in patients with fibromyalgia, low back pain and facial pain (Consoli et al., 2012; O'Brien et al., 2010). Given the link between sleep disturbance and depression (Tsuno, Besset, & Ritchie, 2005), depressive symptoms may modulate pain perception. For example, the association between depression and somatic focus (Mor & Winquist, 2002) is likely to facilitate the perception of pain severity. Exploring their interactions – in both acute and chronic pain states – is necessary to understand their respective roles in developing and maintaining pain conditions. In particular, we need to develop a better understanding of how sleep disturbance is associated with these factors, and how these associations are related to pain outcomes.

Few studies have examined the relationship between sleep disturbance, depressive symptoms, pain-related attention and chronic pain. This study aims to explore the relationships between these parameters and pain severity. We hypothesised that those reporting greater sleep disturbance would experience higher levels of depressive symptoms

and pain-related attention which in turn, would be associated with greater levels of pain severity.

Methods

Participants

Patients currently undergoing treatment for a chronic pain condition were recruited from a specialist pain clinic at Frenchay Hospital (Bristol, United Kingdom). Ethics approval was obtained from the South West Frenchay Research Ethics Committee.

Insert Table 1 about here.

Questionnaires

Questionnaires administered to the patients examined the following constructs: pain, sleep, depressive symptoms and attention to pain. Questionnaire booklets were provided by clinic staff to patients prior to their specialist appointment. Those patients who completed the questionnaires (and thus provided consent) either returned their booklets to clinic staff or were invited to return them via post.

Pain. Pain was assessed using the Brief Pain Inventory (BPI) (Cleeland & Ryan, 1994). The BPI captures how pain interferes with various aspects of life (e.g., general activity, mood, work and relationships) as well as level of pain severity. Only the pain severity domain of the BPI was used in the present analyses. Higher scores indicate more severe pain. Responders rate their pain severity on a scale from 0 (no pain) to 10. The BPI has been validated in clinical samples and has acceptable reliability (Tan, Jensen, Thornby, & Shanti, 2004).

Sleep. Sleep was assessed using the Pittsburgh Sleep Quality Index (PSQI). Using 19 items, participants rate their sleep quality and disturbance over the previous month (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). A validated three-factor structure assesses 'sleep efficiency', 'perceived sleep quality' and 'daily disturbance' (Cole et al, 2006). The three-dimensional structure allows us to better understand the type and nature of sleep disturbance underlying particular clinical symptoms. The 'sleep efficiency' factor corresponds to sleep duration and habitual sleep efficiency. Specifically, this domain allows us to establish the degree of consolidated sleep by assessing the amount of time spent in bed, in comparison to time spent asleep across an individual's regular sleep pattern. 'Perceived sleep quality' takes into account subjective sleep quality, sleep onset latency and sleep-related medication use. Poor sleepers typically have negative perception of their sleep quality and take longer to fall asleep (sleep onset latency of > 15 minutes), which in turn may lead to the use of sleep promoting medications. Finally, 'daily disturbance' relates to both nocturnal (e.g. number of wakes) and daytime disturbances (e.g. napping). The reliability of the three-factor structure has been favourable across a number of clinical samples (Marimann et al, 2012; Nicassio et al, 2015). A global score, ranging from 0-21 can also be attained, with higher scores reflective of worse sleep outcomes. A global score of 8 or above is indicative of a 'poor sleeper' (Carpenter & Andrykowski, 1998). Overall, the PSQI has good internal consistency, construct validity and a specificity of 87% in distinguishing between poor and good sleepers (Buysse et al., 1989).

Depression. The Patient Health Questionnaire (PHQ-9) was used to assess depression (Kroenke, Spitzer, & Williams, 2001). For each PHQ-9 item, participants are asked how often they have experienced certain depressive symptoms over the previous two weeks. Each item has four response options ranging from "not at all" to "nearly every day". PHQ-9 scores range from 0 to 27 and are categorised as: none (0 - 4), mild (5 - 9 points), moderate (10 - 14

points), moderately severe (15 - 19) and severe (> 20 points) depressive symptom severity. The PHQ-9 has been validated in pain samples (Richardson & Richards, 2008).

Pain-related awareness and vigilance. Attention to pain was assessed using the Pain Vigilance and Awareness Questionnaire (PVAQ), a self-report measure consisting of 16 items, each rated on a 6 point scale (0 – "never", 5 – "always") (McCracken, 1997; Roelofs, Peters, McCracken, & Vlaeyen, 2003). A global score can be calculated (0-80), with higher scores indicative of a heightened state of vigilance and attention to pain. The PVAQ has been validated for use in both clinical and non-clinical samples (McCracken, 1997; Roelofs et al., 2003)

Statistical Analyses

Hierarchical linear regression analyses were used to assess the relationship between pain, sleep disturbance, depressive symptoms and attention to pain. Specifically, theoretically-guided block entry was utilised to examine the potential mediating effects of attention to pain and mood (O'Brien et al., 2010). The daily disturbance component of the PSQI, which takes into account nocturnal sleep disruption and daytime dysfunction, was used to explore the association between sleep disturbance and these variables. Pain severity was assessed using the relevant sub-scale of the BPI. Multicollinearity was tested throughout and found to be acceptable.

Structural equation modelling (SEM) allows us to test relationships between observed and latent constructs. It also evaluates the direct and indirect effects among latent variables. The method was chosen to explore potential mechanisms between our variables. SEM was conducted using AMOS 19.0 (Arbuckle, 2010) with maximum likelihood estimation. Full details of the methodologies used are available upon request.

SEM models using only two indicators per latent variable are more susceptible to unreliable error estimates, and are more likely to be under-identified. Three or more indicators are therefore deemed appropriate (Kenny, 2013). Latent constructs of the observed data were built using all of the items included within each questionnaire, respectively. Items were apportioned using random pattern sequences (e.g., n+3) to parcel our observed variables. Depressive symptoms consisted of three indicators (PHQ1: items Q1, Q4, Q7; PHQ 2: Q2, Q5, Q8; PHQ3: Q3, Q6, Q9). Attention to pain (PVAQ1: items Q1, 5, 9, 13; PVAQ2: Q2, Q6, Q10, Q14; PVAQ3: Q3, Q7, Q11, Q15; PVAQ4: Q4, Q8, Q13, Q16). Sleep disturbance consisted of 3 indicators (PSQI1: items7a, 7b, 7c; PSQI2: items 7d, 7e, 7f; PSQI3: items7d, 7g, 4). Internal reliability was acceptable for each (Cronbach's $\alpha = 0.69$ to 0.89). Our initial structural model was based on hypothesised theoretical relationship between our latent variables. Before the structural model was tested, confirmatory factor analysis was used to examine the measurement model which indicated that the sub-scales used were valid operationalisations of the latent constructs used (χ^2 [71] = 104.46, p = 0.06, RMSEA = 0.046, GFI = 0.94, AGFI = 0.91, CFI = 0.98, Bollen-Stein, p = 0.08).

Results

Demographic Information

A total of 242 patients were provided with information about the study and invited to take part in the questionnaire study by clinic staff. Fourteen patients failed to return their questionnaires and a further 7 failed to complete their questionnaires. Therefore, data were available on 221 participants aged between 20 and 84 years (mean = 51, SD = 15), of whom 131 (59%) were female and 208 (94%) were of European ancestry. The mean pain severity

(on a scale of 0 - 10) was 6 (SD = 2) and 85 (39%) had suffered with pain for 10 or more years. Full demographic information is presented in Table 1.

Over 86% (n = 191) of the sample were evaluated as poor sleepers (PSQI > 8) and around 57% took medication specifically for sleep problems. Mann-Whitney U tests indicated that poor sleepers had more depressive symptoms ($z = 4.60$, $p < 0.001$), greater attention to pain ($z = 2.28$, $p < 0.05$) and higher degrees of pain severity ($z = 3.59$, $p < 0.001$). Finally, 56% of patients reached the threshold for clinical depression (PHQ > 10).

Pearson's bivariate correlations were assessed between each of the variables. Pain severity was positively correlated with each of the variables, indicative of an association between heightened pain states and increases in these co-morbidities. Depressive symptoms also shared strong correlations with sleep disturbance and global sleep impairments. All other variables were moderately correlated with each other (Table 2).

Insert Table 2 about here.

Regression Analyses

Hierarchical regression was used to explore the associations between sleep disturbance, depressive symptoms and attention pain in predicting pain severity. Sociodemographic variables were controlled for by entering age and sex into the first step of the analysis. In the second step, pain duration was entered. Daily disturbance was entered and controlled for in the third step. Attention to pain was entered into the fourth step. Finally depressive symptoms were entered into the last, fifth step.

Daily disturbance contributed to the model (*step 3:* $R^2 = 0.14$, $F [1, 216] = 8.76$, $p < 0.001$) until depressive symptoms was entered (Table 3). The final model was found to

predict 28% of the variance in pain severity (R^2= 0.28, F [1, 214] = 14, p <0.001). Both depressive symptoms (β = 0.29, 95% CI: 0.13 to 0.45, t = 3.60, p < 0.001) and attention to pain (β = 0.27, 95% CI: 0.14 to 0.41, t = 4.00, p < 0.001) contributed to the overall model. The addition of depressive symptoms (*step 5*) to the model, reduced the contribution of attention to pain and, importantly, caused sleep disturbance to no longer predict pain severity (β = 0.07, 95% CI: -0.08 to 0.22 t = 0.89, p = 0.37). Overall, results suggest that both depressive symptoms and attention to pain play a mediating role in the relationship between sleep disturbance and pain severity. SEM was conducted to explore possible mechanisms of these interactions.

Insert Tables 3 and 4 about here.

Structural Equation Modelling

The initial model tested the indirect effects of both depressive symptoms and attention to pain on pain severity. In line with recommendations from Preacher and Hayes (2008), the residuals of our two mediators were correlated to account for the fact we would not expect sleep to fully explain the observed association between depressive symptoms and attention to pain. This model (Figure 2) provided a good fit and analysis of the path coefficients indicated that no further model modification was required (all path estimates p < 0.05) (χ^2 [72] = 107.67, p = 0.004, GFI = 0.94, AGFI = 0.91, CFI = 0.98, RMSEA = 0.05, Bollen-Stein, p = 0.065). Sleep disturbance has a strong positive association with depressive symptoms (β = 0.67, 95% CI: 0.57 to 0.75, p < 0.001), which in turn has a moderate positive association with pain severity (β = 0.36, 95% CI: 0.19 to 0.51, p < 0.001). Similarly, sleep disturbance also has a strong association with attention to pain (β = 0.57, 95% CI: 0.45 to 0.67, p < 0.001),

which also had a weak association with pain severity (β = 0.27, 95% CI: 0.11 to 0.42, p < 0.001). The indirect effects were quantified by multiplying the parameter estimates along each path and bootstrapping was used to derive standard errors for these product terms. Whilst the indirect effect through depressive symptoms appeared slightly larger in magnitude (β = 0.24; SE = 0.052) compared to that through attention to pain (β = 0.15; SE = 0.05) there was little evidence (p = 0.228) to suggest that one pathway be of greater magnitude in this model. These final calculations were performed using Mplus version 7.3.

Insert Figure 1 about here.

Discussion

These findings support previous reports of a high prevalence of sleep disorders in chronic pain populations. Using the PSQI, 86% of our participants were assessed as being 'poor sleepers' and reported greater attention to pain, depressive symptoms scores and heightened pain severity. The majority of patients reported that pain caused a high degree of disruption throughout the night and few felt that their sleep was of good quality. Sleep disturbance was associated with increased depressive symptoms in the sample, and to heightened attention to pain stimuli, but was not directly associated with pain severity. Instead, the relationship between sleep disturbance and heightened pain severity appeared to be mediated by depressive symptoms and maladaptive attention processes (such as increased vigilance to pain stimuli). Our results suggest that these processes might play a role in the association between sleep disturbance and pain severity in chronic pain patients. Developing an understanding of how these processes mediate the association between sleep disturbance

and pain could be vital in reducing the chronicity of pain states, and in developing treatment options for patients.

Sleep disturbance measured in this sample considered both nocturnal and daytime disturbances. In addition to difficulty sleeping throughout the night, patients who scored highly on this component reported increased difficulties with social engagements, disturbed daily routines, blunted enthusiasm and daytime hypersomnolence. This compliments previous studies showing that chronic pain patients were found to engage in more physical activity following a better night sleep (Tang & Sanborn, 2014). Reduced engagement with day-time activities can lead patients to isolate themselves. This in turn, can cause negative ruminative cycles regarding one's pain and contribute to the development of depressive symptoms. Sleep disruption itself is commonly associated with negative mood states (Tsuno et al., 2005). Whereas any form of sleep disturbance is likely to lead to lower mood, prolonged disturbance may also lead to symptoms of clinical depression (Franzen & Buysse, 2008; Tsuno et al., 2005). Many of our sampled patients showed PHQ-9 scores indicative of clinical depression; such levels of depression could drive elements of somatic and self-focused attention. Increased somatic preoccupation can lead patients to monitor their body for physiological changes in pain symptoms. Some patients may even focus and ruminate over particular symptoms, making it difficult to escape from pain.

Once dysfunctional affective and attentional processes are in place, these might be maintained by the presence of depressive symptoms and poor coping strategies (Hassett, Cone, Patella, & Sigal, 2000). Indeed, our data would suggest that both heightened attention to pain and depressive symptoms are associated with maintaining the relationship between sleep disturbance and perceived pain severity. Both may therefore play a role in predicting disease chronicity. Further work assessing the longitudinal relationships between these parameters is required to elucidate the potential mechanisms.

Distraction from ruminative thoughts is considered a useful strategy in coping with cycles of negative mood. However, if cognitive flexibility is reduced by sleep disturbance the behavioural response to shifts one's attention away from the threat of pain stimuli is somewhat compromised. Pain will become more apparent in one's consciousness. Given that distraction has analgesic benefits (Johnson, 2005), this the development of dysfunctional attentional and affective processes may increase disease burden. Challenging such maladaptive thought processes, emotions and behaviours would be of key therapeutic benefit to patients. Indeed the use of cognitive behavioural therapy (CBT) in chronic pain has been highly favourable (Morley, Eccleston, & Williams, 1999; Turner, Holtzman, & Mancl, 2007). Recent evidence would support the use of CBT for insomnia (CBTi) in chronic pain. A randomized control study found CBTi to be effective in reducing sleep problems, in addition to alleviating pain-related disability and improving mood (Currie, Wilson, & Curran, 2002). Similar results were also found in fibromyalgia patients (Edinger, Wohlgemuth, Krystal, & Rice, 2005). Improving sleep disturbance might indirectly improve pain severity levels across patients by reducing patient's depressive symptoms and attentional disturbances. This will also facilitate coping mechanisms for dealing with maladaptive sleep-related cognitions to promote sleep. Pain management strategies should include both pharmacological and psychological interventions to promote both physical and cognitive rehabilitation.

Although this study indicates some interesting associations, there are limitations to the interpretation of the results. First, the self-report nature of the data means that the results must be interpreted with caution. There may be a tendency to over- or under-report symptoms, especially when one considers the private and subjective nature of pain. Second, whereas parcelling is useful in reducing problems of model estimation, convergence and stability, there are some disadvantages associated with the approach. Improper parcelling can result in issues with both model misspecification and interpretation. Nonetheless, parcelling confers a

number of empirical and psychometric advantages, and ultimately allows SEM approaches to be used. A full discussion of the advantages and disadvantages of parcelling is provided by Little et al (2013). Third, the data are cross-sectional and correlational, meaning we cannot infer causality from our data. Importantly, we cannot draw firm conclusions about mediation effects. Both longitudinal measures and experimental manipulations are needed to explore the interactions between sleep, pain and other co-morbid factors. Such measures could elucidate the role of such interactions in both the development and progression of pain states. Fourth, data were not collected on specific pain diagnoses. Differing underlying condition pathogeneses could play a role in the relationship between pain and sleep; therefore, in future it would be of use to collect such data to allow for sub-group analyses. Fifth, information on medication was not collected and as such could not be controlled for within our analyses. Problematically, certain analgesics compounds can cause sleep fragmentation, and even promote insomnia (Furlan, Sandoval, Mailis-Gagnon, & Tunks, 2006). Future studies should consider the influence medication might be having on both sleep architecture and both affective and somatic states.

Finally, it is possible to fit a number of equivalent, or near-equivalent models. Equivalent models are possible, simply by revising configuration of the paths included. Such equivalent models may be predictive of the same correlations and co-variances may arise, in addition to having equal fit statistics and approximate fit indices (Stelzl, 1986; Lee and Hershberger, 1990). The importance of models that generate slightly difference co-variances, or near-equivalence, must also be considered. Furthermore, when presented with a complex phenomenon, competing theories may give rise to a number of alterative non-equivalent models. For example, fitting equivalent or near-equivalent models in the present data may allow us to better explore the role of depression in the relationship shared by sleep and pain. Such results could further reinforce studies indicating that depression has a mediating effect

on sleep and pain (Hamilton et al., 2012; Miró, Martínez, Sánchez, Prados, & Medina, 2011; O'Brien et al., 2010).

If these models fit are of comparable fit, theoretical justification should be provided conveying why one particular model is preferred over a mathematically identical one. We believe our current model is built on a solid theoretical framework and is consistent with our data. Using such a hypothesis driven test of the data allowed us to explore some novel associations that may help further our understanding of processes underlying the maintenance of chronic pain states. Nonetheless, such models need to be tested in other samples before we can make any strict conclusions on the associations observed.

Our findings indicate the potentially critical role of depressive symptoms and attentional systems in the relationship between sleep disruption and pain severity. Broadening treatment options to consider these co-morbid factors can reduce the reliance upon pharmacological interventions. This will allow patients to better understand and cope with their condition and if treated soon enough, can assist in preventing the development of chronicity in certain patients.

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Conflicts of Interest

The authors declare that there are no relevant conflicts of interest. Each author contributed significantly to the design, analysis and preparation of the manuscript. All have approved the final version of the manuscript.

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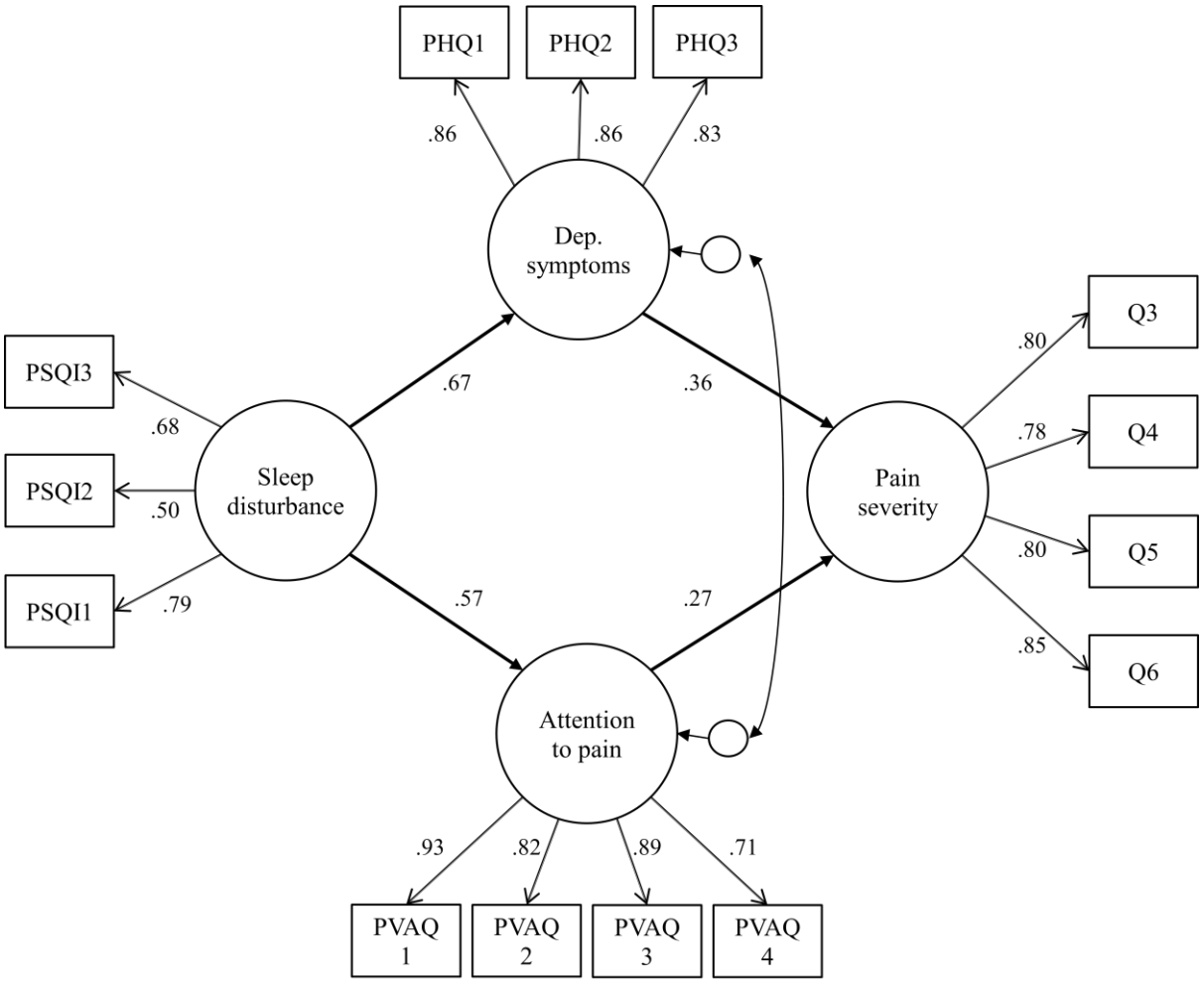

Figure 1: Final structural equation model for the relationship between sleep disturbance, depressive symptoms, attention to pain and pain severity. Standardised estimates are indicated above path estimates.

Table 1. Demographic information for the patients (n = 221)

Age	Mean = 51 (SD = 15)	Range = 20 - 84
		N (%)
Gender	Female	131 (59)
Ethnicity	White	208 (94)
	Black	4(2)
	Asian	6 (3)
	Other	3 (1)
Pain Duration	0 - 6months	1 (1)
	6-12 months	6 (3)
	1 to 2 years	21 (10)
	2-3 years	25 (11)
	3-4 years	21 (10)
	4-5 years	11 (5)
	5-10 years	51 (23)
	10 or more years	85 (39)
Work Status	Full time	42 (19)
	Part time	37 (17)
	Unemployed	12 (5)
	Retired	62 (28)
	Student	2 (1)
	Unable to work	66 (30)
Pain Severity Score	Mean = 5.9 (SD = 1.8)	
Pain Interference Score	Mean = 6.2 (SD = 2.1)	

Table 2. Descriptive statistics and Pearson's bivariate correlations between the variables**.**

	Mean (SD)	**Range**	**2**	**3**	**4**	**5**	**6**	**7**
1. BPI Severity Score	5.89 (1.8)	0 - 10	0.42	0.43	0.34	0.39	0.25	0.43
2. Pain-related Awareness (PVAQ)	52.35 (12.4)	0 - 80	#	0.49	0.42	0.33	0.20	0.39
3. Depressive Symptoms (PHQ-9)	11.53 (6.7)	0 - 27		#	0.63	0.50	0.35	0.61
4. PSQI Disturbance	3.46 (1.1)	0 - 6			#	0.42	0.3	0.63
5. PSQI Perceived Quality	5.32 (2.4)	0 - 9				#	0.38	0.85
6. PSQI Efficiency	3.38 (2)	0 - 6					#	0.76
7. PSQI Global	12.1 (4.2)	0 - 21						#

BPI denotes the Brief Pain Inventory; PVAQ, Pain Related Vigilance and Awareness Questionnaire; PHQ-9, Patient Health Questionnaire; PSQI, Pittsburgh Sleep Quality Index. All correlations $p<0.05$

Table 3. Overall model fit and changes in analyses predicting pain severity.

	R	***R²***	**a*R²***	**SE**	***R²* Change**	***F***	***P***
Age and Gender	0.10	0.01	0.01	1.78	0.01	0.99	0.373
+Duration	0.13	0.02	0.01	1.78	0.01	1.28	0.282
+PSQI Disturbance	0.37	0.14	0.12	1.66	0.12	8.76	< 0.001
+Pain-related Awareness (PVAQ)	0.49	0.24	0.22	1.57	0.10	13.45	< 0.001
+Depressive Symptoms (PHQ-9)	0.53	0.28	0.26	1.53	0.04	13.99	< 0.001

Table 4: Predictors of pain severity in the final regression equation.

	Variables	*B*	SE	β	P-value
Step 1	Age	0.01	0.01	0.04	0.56
	Gender	-0.31	0.24	-0.08	0.21
Step 2	Age	0.00	0.01	0.03	0.68
	Gender	-0.32	0.24	-0.09	0.19
	Duration	0.09	0.06	0.09	0.18
Step 3	Age	0.01	0.01	0.08	0.22
	Gender	-0.34	0.23	-0.09	0.14
	Duration	0.76	0.06	0.08	0.20
	PSQI	0.57	0.10	0.35	<0.001
	Disturbance				
Step 4	Age	0.01	0.01	0.12	0.05
	Gender	-0.24	0.22	-0.07	0.26
	Duration	0.06	0.06	0.07	0.27
	PSQI	0.35	0.11	0.21	0.001
	Disturbance				
	PVAQ	0.05	0.01	0.35	<0.001
Step 5	Age	0.02	0.01	0.14	0.02
	Gender	-0.34	0.21	-0.10	0.11
	Duration	0.45	0.06	0.05	0.41
	PSQI	0.11	0.12	0.07	0.37
	Disturbance				
	PVAQ	0.04	0.01	0.27	<0.001
	PHQ-9	0.08	0.02	0.3	<0.001

PVAQ denotes Pain Related Vigilance and Awareness Questionnaire; PHQ-9, Patient Health Questionnaire