

Self-efficacy beliefs mediate the association between pain intensity and pain interference in acute/subacute whiplash-associated disorders

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**SELF-EFFICACY BELIEFS MEDIATE THE ASSOCIATION BETWEEN PAIN
INTENSITY AND PAIN INTERFERENCE IN ACUTE/SUBACUTE
WHIPLASH-ASSOCIATED DISORDERS**

Abstract

Purpose: To evaluate whether a set of pre-accident, accident related, post-accident treatment and psychosocial factors mediate the association between pain intensity and: (1) pain interference; and (2) expectations of recovery in individuals with acute/subacute whiplash-associated disorders (WAD). We also aim to explore the potential mediating pathways (if any) within different psychosocial factors.

Methods: This was a cross-sectional study conducted on a sample of 173 participants with acute/subacute WAD. Bayesian Network (BN) analysis was used to understand the probabilistic dependency relationships between a set of pre-accident, accident related, post-accident treatment, pain, and psychosocial (pain interference, pessimism, expectations of recovery, pain catastrophizing, and self-efficacy beliefs) variables.

Results: The results revealed that self-efficacy beliefs partially mediated the association between pain intensity and pain interference. Self-efficacy beliefs partially mediated the association between pain intensity and pain catastrophizing whereas kinesiophobia partially mediated the association between self-efficacy and pain catastrophizing. Psychological factors did not mediate the association between pain intensity and expectations of recovery.

Conclusion: These results indicate that individuals with acute/subacute WAD may present with lesser pain interference and pain catastrophizing associated with a determined pain intensity value when they show greater self-efficacy beliefs. As the cross-sectional nature of this study limits firm conclusions on the causal impact,

researchers are encouraged to investigate the role that patient's self-efficacy beliefs play in the transition to chronic WAD via longitudinal study designs.

Keywords: whiplash; acute; observational study; psychological factors; neck pain

Introduction

Whiplash-associated disorders (WAD) remain a challenge to manage [1]; they affect up to one million people each year in the United States alone [2] and the socio-economic cost of this disorder is substantial [3]. Neck pain, pain interference, post-traumatic stress disorders, and sleep difficulties are common among individuals with WAD [4, 5]. Disturbances in motor and sensory function, as well as cognitive processing alterations, are also frequently observed [6, 7]. Although a rapid improvement may occur during the first three months following a whiplash injury, recovery rates typically then stabilise [8]. Numerous biopsychosocial factors including widespread pain, sensorimotor incongruence, social withdrawal, and depressed mood may be involved in the persistence of pain and pain interference following a whiplash injury [9]. Theoretical pain models have postulated that negative psychological factors such as pain catastrophizing, fear of pain/movement and pain-related anxiety foster the use of passive coping strategies in response to pain e.g. avoidance behaviours or pain hypervigilance [9–11]. This may be associated with a worse prognosis following a whiplash injury [9–11]. In this sense, a recent systematic review has found that some negative psychological factors including poor expectations of recovery, post-traumatic stress symptoms, and passive coping strategies may increase the risk of developing chronic neck pain and/or disability [12]. On the other hand, previous cross-sectional studies have revealed that self-efficacy beliefs can be protective in people with acute WAD [13, 14].

Previous research has evaluated the mediating effects of some psychological factors in the association between pain and pain-related outcomes (e.g. pain interference) in individuals with WAD before the onset of chronicity. For example, self-efficacy beliefs have been shown to mediate the association between pain intensity and pain interference in a cross-sectional study [14]. Pain catastrophizing and fear of movement have been reported to mediate the association between pain intensity and disability [13]. Additionally, longitudinal data have reported that fear of movement, but not pain catastrophizing, mediated the association between pain intensity and disability [15]. However, neither control over pain nor the ability to reduce pain mediated the association between functional self-efficacy or pain catastrophizing and pain interference in a longitudinal study [13]. To the best of our knowledge, the simultaneous mediating effects of multiple psychological factors between pain intensity and pain interference in acute or subacute WAD has not been evaluated. This information would allow us to compare the relative importance of different psychological factors as mediators in these associations.

The current study had three objectives using Bayesian Networks (BN). In people with acute/subacute WAD, this cross-sectional study sought to evaluate whether a set of pre-accident demographic, accident related, post-accident treatment and psychosocial factors mediate the association between pain intensity and: (1) pain interference; and (2) expectations of recovery. We hypothesised that all psychological factors mediate the association between pain intensity and the aforementioned pain-related outcomes (pain interference and expectations of recovery). The last objective of this study was to explore the potential mediating pathways (if any) within different psychosocial factors.

Methods

Study design

This cross-sectional study was conducted between August 2018 and September 2019 at a private Physiotherapy clinic in Malaga, Spain. This study followed the Declaration of Helsinki and is reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) criteria [16]. The study was approved by the Ethics Committee and Research of Malaga, Spain (13122018). Participants provided informed written consent.

Participants and Setting

Participants attending the Physiotherapy clinic to receive treatment for their WAD were invited to participate in this study. A physiotherapist with 10 years of expertise in the management of musculoskeletal pain disorders, screened potential participants for eligibility. Inclusion criteria were adults > 18 years with acute or subacute WAD (pain duration less than 3 months from the onset of injury) attributed to a motor vehicle accident. We excluded individuals with an inability to complete patient-reported outcome measures. A convenience sample of 178 participants with acute or subacute WAD was recruited, however, five patients did not provide all necessary information and thus, a final sample of 173 participants was considered for the analysis.

Variables

The following 16 variables were collected and grouped into five categories to build the BN:

1: Pre-accident variables:

- Age: age (years) of the participant upon study enrolment.
- Sex: male or female.
- Educational level coded as (i) university \geq four years; (ii) university up to four years; (iii) high school; (iv) school; (v) no formal education.

- Employment coded as: (i) unemployed; (ii) freelance; (iii) employee.

2: Accident related variables

- Type of vehicle coded as: (i) car; (ii) motorbike; (iii) bike; (iv) bus; (v) others.
- Passenger's position in the vehicle coded as (i) driver; (ii) co-pilot; (iii) back seat.

3: Post-accident pain characteristic variables

- Current pain intensity: a visual analogue scale (VAS), where zero implies no pain and 10 the worst pain imaginable, was used to evaluate pain intensity. This tool is valid and reliable to assess pain intensity [17].
- Pain duration is based on weeks from the accident.

4: Post-accident psychosocial and treatment variables

- Current treatment coded as: (i) no treatment; (ii) medication + physiotherapy; (iii) injections; (iv) physiotherapy; (v) other treatments (massage, reflexology, acupuncture).
- Self-efficacy beliefs: the Spanish version of the Chronic Disease Self-Efficacy (CDSE) (internal consistency Cronbach's α 0.85 and a test-retest validity 0.80) was used [18]. This tool consists of four items whose score ranges from 0 "very insecure" to 10 "very safe". The total score can range from 0 to 40, with higher scores reflecting greater self-efficacy beliefs.
- Pessimism: the Spanish version of the Life Orientation Test-Revised (LOT-R) (internal consistency Cronbach's α =0.90 [19]; test-retest reliability=0.72 [20]) (pessimism subscale) was used [21]. This tool is composed of three items

whose score ranges from 0 “strongly disagree” to 4 “strongly agree”. The total score can range from 0 to 12, with higher scores reflecting greater pessimism.

- Kinesiophobia: the Spanish version of the Tampa Scale for Kinesiophobia short-form (TSK-11) (internal consistency Cronbach's α 0.79) was used [22, 23]. This tool is composed of 11 items whose score can range from 1 “strongly disagree” to 4 “completely agree”. The total score can range from 11 to 44, with higher scores reflecting greater kinesiophobia.
- Pain catastrophizing: the Spanish version of the Pain Catastrophizing Scale (PCS) (internal consistency Cronbach's α 0.79; test-retest reliability 0.84) was used [24]. This tool is composed of 13 items, whose score can range from 0 “not at all” to 4 “all the time”. The total score can range from 0 to 52, with higher scores reflecting greater pain catastrophizing.
- Sick leave: coded as: (i) no; (ii) retired; (iii) yes.

5: Outcome variables

- Recovery expectations: A single question “What chance of recovery do you think you will have once you finish the treatment?” was used which was scored on an 11-point Likert scale, where zero means no chance of recovery and 10 means total recovery. Greater scores reflect higher levels of positive expectations of recovery.
- Pain interference: the Spanish version of the Neck Disability Index (NDI) (internal consistency Cronbach's α 0.89; intra-class correlation coefficient 0.98) was used [25]. This tool is composed of 10 items, each ranging from 0 to 5 [25]. Greater scores reflect higher levels of pain interference (disability).

Approach to data analysis

All the BN analyses were conducted in R software using the BN learn package [26]. The codes can be found in the supplementary material (appendix A). BN quantifies the relationships among a set of variables $X = \{X_1, \dots, X_N\}$, where N is the number of variables, using a directed acyclic graph (DAG). Each variable is associated with a node and directed arcs represent conditional dependencies between pairs of nodes. Building a BN model using a data-driven approach involves two stages: 1) structural learning - identifying which arcs are present in the DAG; and 2) parameter learning - estimating the parameters that regulate the strength and the direction of the corresponding relationships. In other words, the BN structure gives the (putative) causal direction and the parameters give the magnitude and the sign of the relationship (i.e. positive vs negative relationship, etc.).

We made use of blacklisting and model averaging to reduce the number of arcs that are incorrectly included in the BN. A blacklist is simply a set of relationships that we know are less likely to exist, as they go against known biological/physical mechanisms, and are ignored during structural learning. We blacklisted arcs that pointed from a higher category number to a lower category number. For example, a person's education level would not likely affect the position within the vehicle during the accident. All blacklisted arcs included are found in Appendix B. Model averaging consists of resampling the data multiple times ($B = 200$) using bootstrap and performing structure learning on each of the resulting samples; in the following, we will use the hill-climbing (HC) algorithm for this purpose. We computed an "average" consensus DAG by selecting those arcs that have a frequency of $>50\%$ in the bootstrapped samples, to create a sparse and interpretable network [27].

Bayesian networks (BN) can easily incorporate prior knowledge available from the literature and expert opinions into the models, by encoding prior knowledge in sets of

whitelisted arcs. We built a second BN model using the same blacklists as the first model but added dependent relationships reported in the literature (Table 1) [14, 15, 28].

The second BN model was used to compare its predictive correlation with the first model (without a whitelist). If the empirical data supported the dependent relationships reported in the literature, then the predictive correlation of the second model would be superior to that of the first model. To determine the validity of the model, validation was performed using nested 10-fold cross-validation. Nested 10-fold cross-validation iteratively splits the training set into 10 approximately equal folds, trains the model on 9 folds, evaluate the model's performance on the 10th fold, and averaging the performance metric across all 10 evaluations. The metric of model performance was defined by computing the correlation coefficient between the predicted and observed values of each continuous variable. The strength of correlation was categorized as negligible ($|r| \leq 0.30$), low ($|r| = 0.31$ to 0.50), moderate ($|r| = 0.51$ to 0.70), high ($|r| = 0.71$ to 0.90) and very high ($|r| = 0.91$ to 1) [29].

Conditional probability queries. The derived averaged BN model can be considered an “expert system”, which means that we can elicit a sample of realizations of the modelled variables under specific conditions. For example, we can query the system to infer the values of the NDI when neck pain intensity reduces by a threshold value. For each conditional probability query, we sampled 10^4 realizations of the variables of interest to obtain precise probability estimates. We used a technique known as belief updating, which estimates the posterior probability of an event happening based on the available evidence on the values of certain variables. We adopted a specific method of belief updating known as logic sampling [30].

Results

The descriptive characteristics of the 16 variables used for BN analysis are presented in Table 2. Figures 1 and 2 show the averaged BN consensus model for models 1 and 2, learned from 200 networks constructed from the data, with arcs appearing at least in 50% of the networks kept. We included the predictive correlations for all variables in Table 3. Given that the predictive validity of the two models was similar, we performed conditional probability queries on model 1. An advantage of BN is that the model enables the reader to query the system on any arbitrary set of clinical questions. To this end, we focused on several interesting clinical queries, and present the ensuing results here.

Pathway(s) leading to pain interference

Based on model 1 (Figure 1), two paths were associated with pain interference. From the sampled posterior distribution, a one-point increase in pain intensity resulted in a 2.08-point increase in pain interference ($t = 67.57$, $p < 0.001$) (Figure 3). One pathway was a direct path between pain intensity and pain interference, whilst another was an indirect path passing through self-efficacy beliefs.

We simulated a scenario where self-efficacy beliefs were not dependent on pain intensity, by fixing the value of the self-efficacy beliefs regression coefficient in the local distributions to zero, which is equivalent to removing the pain intensity-self-efficacy beliefs arc. When fixing the value of self-efficacy beliefs to zero (i.e. the only path from pain intensity to pain interference is the direct path), a one-point increase in pain intensity resulted in a 1.54-point increase in pain interference ($t = 56.32$, $p < 0.001$). This result suggests that self-efficacy beliefs mediated $\sim 26\%$ ($\frac{2.08-1.54}{2.08}$) of the total relationship between pain intensity and pain interference.

Pathway(s) leading to expectations of recovery

None of the included psychological factors demonstrated mediating effects in the association between pain intensity and expectations of recovery (Figure 1 and Figure 2).

Pathway(s) leading to pain catastrophizing

Two paths were associated with pain catastrophizing (Figure 1). From the sampled posterior distribution, a one-point increase in self-efficacy beliefs resulted in a -0.69-point decrease in pain catastrophizing ($t = -72.56$, $p < 0.001$) (Figure 4). One pathway was a direct path between self-efficacy beliefs and pain catastrophizing, whilst another was an indirect path passing through kinesiophobia.

When fixing the value of kinesiophobia to zero (i.e. the only path from self-efficacy beliefs to pain catastrophizing is the direct path), a one-point increase in self-efficacy beliefs resulted in a -0.48-point decrease in pain catastrophizing ($t = -53.45$, $p < 0.001$).

This result suggests that kinesiophobia mediated $\sim 13\%$ ($\frac{0.69-0.48}{0.69}$) of the total relationship between self-efficacy beliefs and pain catastrophizing.

Discussion

To our knowledge, this is the first study analysing the role that a large number of psychological factors play as mediators of the association between pain intensity and different pain-related outcomes (expectations of recovery, pain catastrophizing, and pain interference) in the same sample of acute/subacute WAD, using a BN approach. We found that none of the included psychological factors mediated the association between pain intensity and expectations of recovery. Furthermore, self-efficacy beliefs partially mediated the association between pain intensity and pain interference and pain catastrophizing whereas kinesiophobia partially mediated the association between self-efficacy beliefs and pain catastrophizing. The results of the present study suggest that individuals with acute/subacute WAD may present lesser pain interference and pain

catastrophizing associated with a determined pain intensity value when they show greater self-efficacy beliefs.

Self-efficacy is the belief that one can conduct a determined activity/movement and produce the desired effect despite the potential difficulties [31]. This factor is important in the prognosis of different chronic pain conditions [32] and specifically, in chronic musculoskeletal pain [33]. Previous research also suggests that self-efficacy is a potential mediator of the association between pain intensity and disability in individuals with headache, osteoarthritis, and chronic low back pain [34–36]. A cross-sectional investigation of individuals with non-chronic WAD reported that self-efficacy beliefs mediated the association between pain intensity and pain interference [14] which is in agreement with our results. All these findings indicate that people who can elicit greater self-efficacy beliefs in response to an increase in pain intensity may report lower levels of pain interference.

The current study found that self-efficacy beliefs only mediated ~26% of the relationship between pain intensity and pain interference. This suggests that either the influence of pain intensity on pain interference is the dominant factor [37] or that variables (e.g. mental health comorbidity [38] and psychological inflexibility [39]) not included in the present study could mediate this relationship. In a previous systematic review of mediation studies in spinal pain disorders, it was reported that self-efficacy, psychological distress, and fear mediate the pain intensity-interference relationship [40]. Interestingly, psychological distress in the primary studies of the review was measured using the Depression Anxiety and Stress Scale or the Patient Health Questionnaire (depression subscale) [40]. This suggests that depressive and anxiety symptoms, not included in the present study, could mediate in part the relationship between pain intensity and interference, which would be an important area for future investigations.

Considering the association between pain intensity and pain catastrophizing, a surprising finding of the present study was that kinesiophobia preceded pain catastrophizing. This stands in contrast to the fear-avoidance model [41], but the present results have empirical support from the literature. Greater fear may heighten the cognitive mechanisms that result in selective attention to threatening stimuli such as catastrophic thoughts [42] and hypervigilance [43] which from an evolutionary perspective, confer the organism greater survival benefits [44, 45]. Additionally, self-efficacy beliefs showed a direct path towards pain catastrophizing which may indicate the importance of assessing self-efficacy beliefs when physical (e.g. pain interference) and cognitive (e.g. pain catastrophizing) factors are considered among individuals with non-chronic WAD.

Finally, none of the psychological factors showed a mediating effect in the association between pain intensity and expectations of recovery. Although prior research has demonstrated that poor expectations of recovery are prognostic for the development of chronic WAD [12], the evidence incorporating expectations of recovery as an outcome measure for WAD is scarce. A cross-sectional study found that depressive symptoms, as well as a set of factors such as pain-related variables, economic, and sociodemographic factors, were related to poor expectations of recovery [46]. However, the level of depressive symptoms was not assessed in our sample. Future longitudinal studies should evaluate the role that psychological factors play as mediators of the association between pain intensity and expectations of recovery following a whiplash injury as this would enable more robust conclusions.

Clinical considerations

Given that self-efficacy belief partially mediated the association between pain intensity and pain interference and pain catastrophizing, it would be interesting to speculate the clinical implications of such findings. In this sense, low levels of self-efficacy beliefs are

considered a barrier to musculoskeletal care [47]. Individuals with musculoskeletal pain with higher levels of self-efficacy often show greater self-confidence and willingness to take risks in comparison to those with lower self-efficacy [47], which may improve recovery. Therefore, clinicians can enhance a patient's self-efficacy belief by facilitating mastery of experience, vicarious experience, verbal persuasion, and the education of body response [48]. A recent systematic review found that exercise and psychological interventions may be useful therapeutic approaches to increase pain self-efficacy in people with chronic musculoskeletal pain [49]. Moreover, previous randomized controlled trials have found that exercise and cognitive-behavioural interventions may improve self-efficacy beliefs in acute and chronic WAD [50, 51].

Methodological considerations

This study has several strengths. First, our study applied a BN approach by incorporating prior knowledge available from the literature and expert opinion into the models. We found that our model 1 (empirical data) attained similar correlation values to model 2 (created based on previous knowledge) which adds confidence to our conclusions. Second, we recruited a considerable number of participants and evaluated a large number of psychological factors as mediators of the association between pain intensity and pain interference/expectations of recovery.

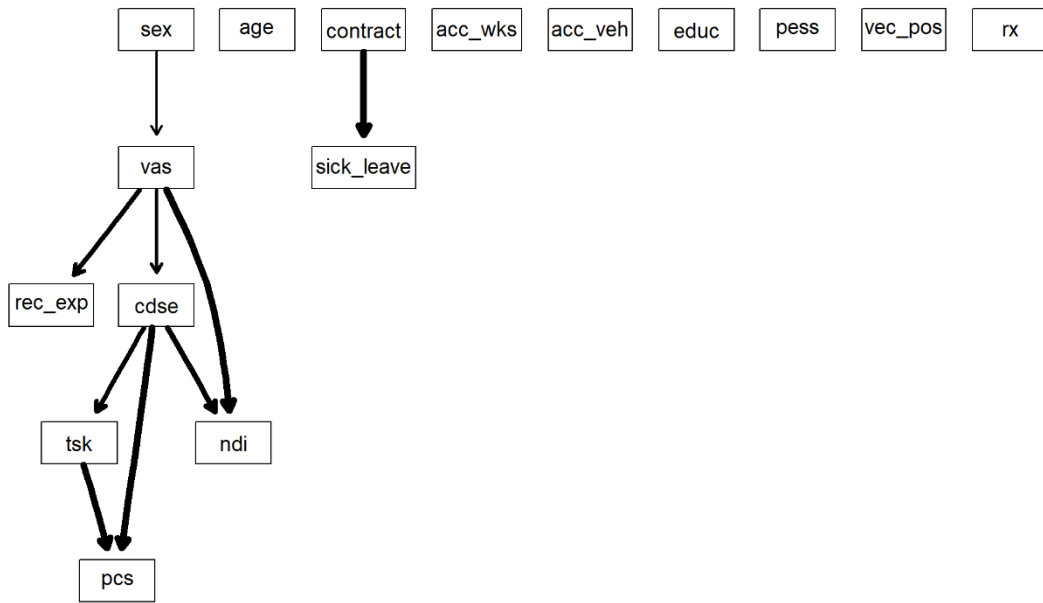
Limitations of the study need to be acknowledged. First, similar to previous mediation research [10, 52, 53] this was a cross-sectional study that limits firm conclusions on the causal nature of the relationships explored in the present study. **Second, the previous psychological – psychiatric, and medication status of the participants were not evaluated. These variables could act as moderator-mediator factors.** Third, the variables included presently were based on prior knowledge about their mediating and prognostic value, and excluded physical variables (e.g. neck muscle endurance [54]). Realistically, the number

of variables included in a BN model must depend not only on prior knowledge but should also consider the logistical feasibility of measuring these measures in a clinical or research environment. However, we view the relationships learned in this study within a “hypothesis-generation” framework, where plausible mediators identified could be targets of intervention in future randomized controlled studies. Future longitudinal and experimental studies are needed.

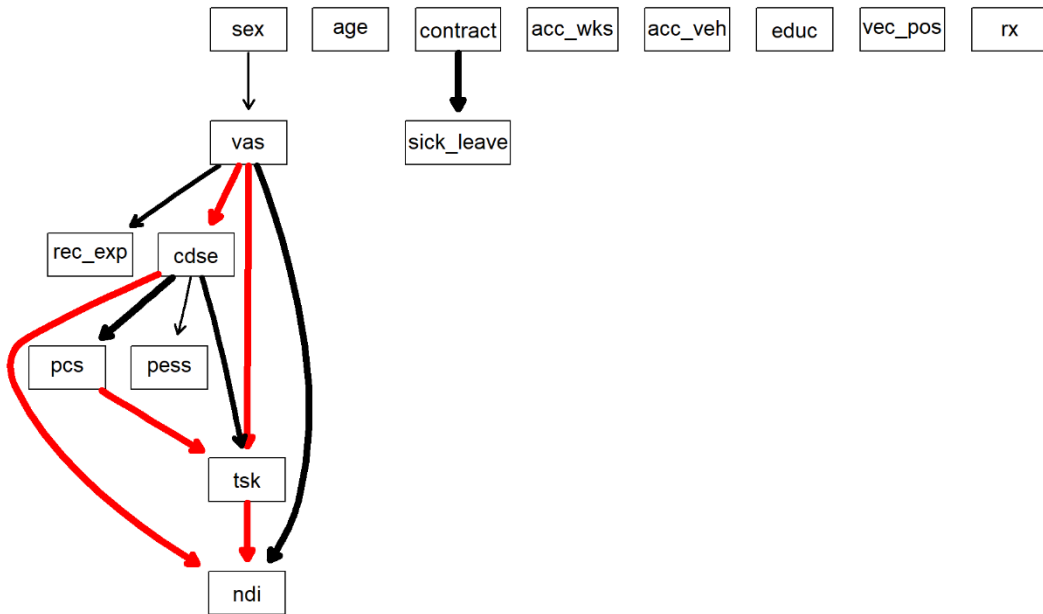
Conclusions

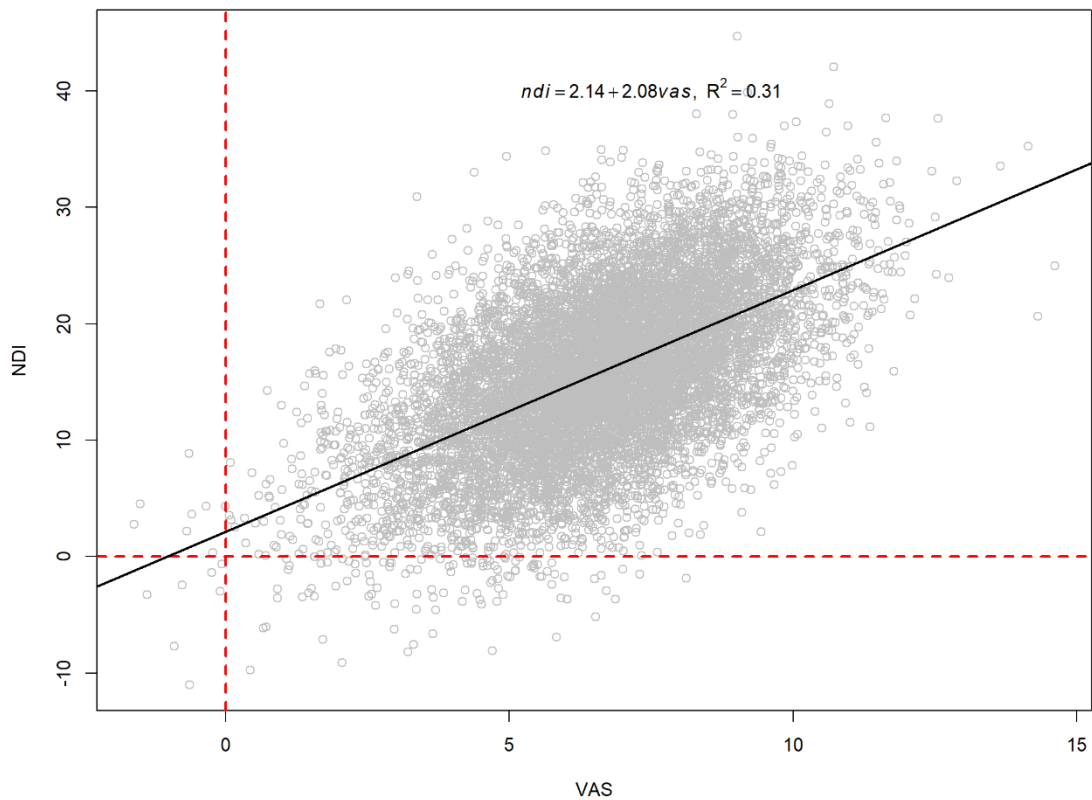
Self-efficacy beliefs partially mediated the association between pain intensity and pain interference and self-efficacy beliefs partially mediated the association between pain intensity and pain catastrophizing whereas kinesiophobia mediated the association between self-efficacy and pain catastrophizing in people with acute/subacute WAD. This indicates that individuals with greater self-efficacy beliefs present with lesser pain interference and pain catastrophizing for given pain intensity. Pain catastrophizing, kinesiophobia, and pessimism did not mediate the association between pain intensity and pain interference. Additionally, no psychological factors mediated the association between pain intensity and expectations of recovery.

Model 1



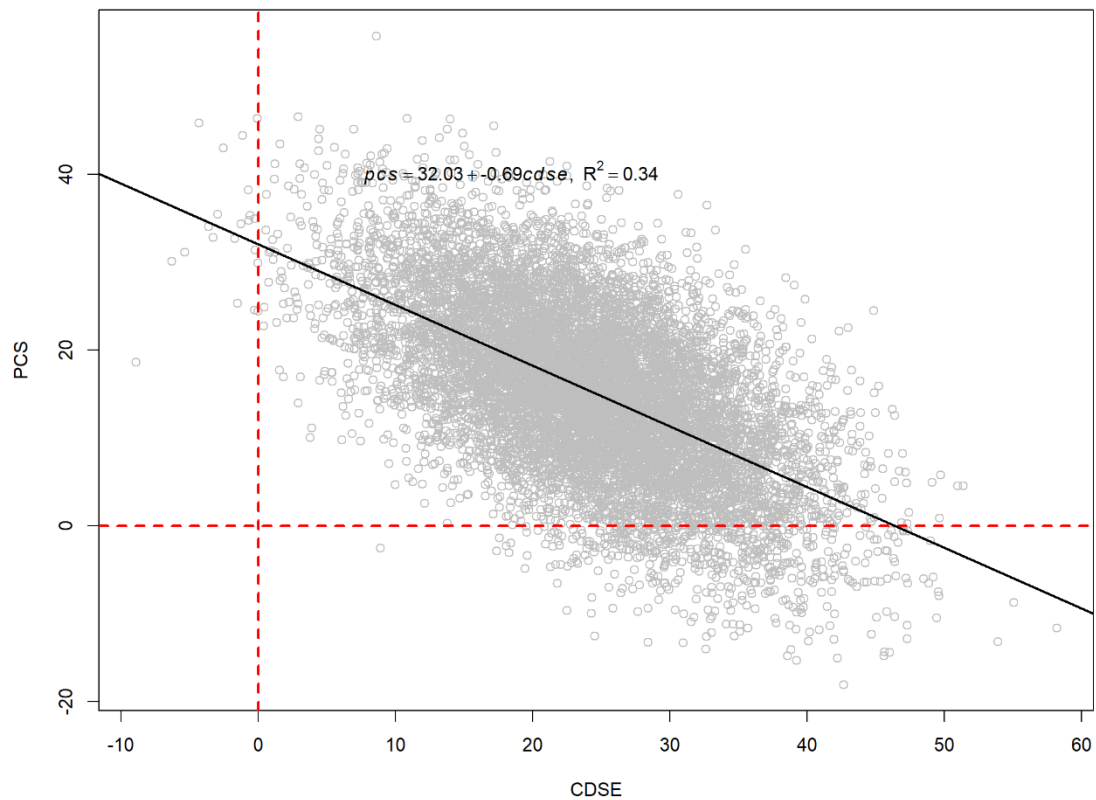
Model 2





Correlation between observed and predicted change values

| Variable | Model | Value | Strength |
|----------------------|-------|-------|------------|
| VAS | No_WL | 0.59 | moderate |
| Recovery Expectation | No_WL | 0.31 | low |
| Pessimism | No_WL | 0.07 | negligible |
| SES | No_WL | 0.68 | moderate |
| TSK | No_WL | 0.57 | moderate |
| PCS | No_WL | 0.60 | moderate |
| NDI | No_WL | 0.66 | moderate |
| VAS | WL | 0.58 | moderate |
| Recovery Expectation | WL | 0.36 | low |
| Pessimism | WL | 0.08 | negligible |
| SES | WL | 0.67 | moderate |
| TSK | WL | 0.58 | moderate |
| PCS | WL | 0.59 | moderate |
| NDI | WL | 0.66 | moderate |



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Figure legends

Figure 1. The directed acyclic graph (DAG) underlying the consensus Bayesian Network of learned from the variables across 173 participants (model 1). The thickness of the arcs is in proportion to their strength. Only arcs with strength > 0.5 are included in the consensus network.

Abbreviations: CDSE: Chronic disease self-efficacy; LOT-R: Life orientation test-revised; NDI: neck disability index; NRS-pain: a numerical rating scale for pain; PCS; pain catastrophizing scale; SD: standard deviation; TSK: Tampa scale of kinesiophobia.

Figure 2. The directed acyclic graph (DAG) underlying the consensus Bayesian Network of learned from the variables across 173 participants (model 2). Arcs in red are enforced to be present in the network by the whitelist. The thickness of the arcs is in proportion to their strength. Only arcs with strength > 0.5 are included in the consensus network.

Abbreviations: CDSE: Chronic disease self-efficacy; LOT-R: Life orientation test-revised; NDI: neck disability index; NRS-pain: a numerical rating scale for pain; PCS; pain catastrophizing scale; SD: standard deviation; TSK: Tampa scale of kinesiophobia.

Figure 3. Increases in pain interference (NDI) for every 1-point increase in pain intensity (VAS) with no variables mediating this relationship.

NDI: neck disability index; VAS: visual analogue scale

Figure 4: Increases in pain interference (NDI) for every 1-point increase in pain intensity (VAS) when self-efficacy mediated this relationship.

NDI: neck disability index; VAS: visual analogue scale

Table legends

Table 1. Whitelist Arcs Used in Second Bayesian Network Model (Using Knowledge from Kamper et al (19), Crombez et al. (8) and Söderlund et al. (42)).

Abbreviations: NDI: the Neck Disability Index; PCS: the Pain Catastrophizing Scale; SES: the Self-Efficacy Scale; TSK: the Tampa Scale for Kinesiophobia; VAS: the visual analogue scale

Table 2. Characteristics of the included participants (n= 173)

Table 3. The predictive correlations for all variables in both models.

Abbreviations: CDSE: the Chronic Disease Self-Efficacy; NDI: the Neck Disability Index; PCS: the Pain Catastrophizing Scale; TSK-11: the Short-Version of the Tampa Scale for Kinesiophobia; VAS: the visual analogue scale.