International Journal of Behavioral Medicine (2019) 26:59–68
https://doi.org/10.1007/s12529-018-9754-8

A Network Analysis of the Links Between Chronic Pain Symptoms and Affective Disorder Symptoms

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Published online: 30 October 2018

Abstract

Background A range of psychological constructs, including perceived pain, self-efficacy, and pain avoidance, have been proposed to account for the comorbidity of chronic pain and affective disorder symptoms. Despite the likely inter-relation among these constructs, few studies have explored these predictors simultaneously. As such, the relative contributions of these psychological influences remain an open question.

Purpose The present study uses a novel, network model approach to help to identify the key psychological contributors to the pain-affective disorder link.

Method A cross-sectional design was implemented. The sample comprised 169 individuals with chronic pain (M_{age} 49.82; range 22–80 years; 58% female) admitted to a metropolitan chronic pain clinic in Victoria, Australia. Participants completed self-report measures of anxiety, depressive, and pain symptoms, pain self-efficacy, fear avoidance beliefs, perceived control, and pain-related disability.

Results Network analysis identified self-efficacy, fear avoidance, and perceived disability as key constructs in the relationship between pain and affective disorder symptoms, albeit in different ways. While self-efficacy appeared to have direct links to other constructs in the network model, fear avoidance and perceived disability seemed to function more as mediators, linking other constructs in the model. Perceived control and anxiety were found to be less influential in the model.

Conclusions Present findings identify self-efficacy, fear avoidance, and perceived disability as plausible candidate variables to target to disrupt the link between pain experience and affective disorder symptoms. However, further testing with longitudinal designs is needed to confirm this.

Keywords Pain · Cognitive factors · Depression · Anxiety · Fear-avoidance · Network analysis

Introduction

Depression and anxiety are common mental health problems with elevated prevalence rates among chronic pain sufferers relative the general population [1, 2]. This relationship between chronic pain and affective disorder symptoms has been shown to be bi-directional [e.g. 3–5], and underpinned by a variety of psychological mechanisms influencing the way an individual processes pain-related information and perceives her/his capacity to manage their condition [6–9]. For instance, Wong and colleagues [9] found that the relationship between negative emotions and adjustment to pain symptoms was mediated via catastrophizing and pain-related fear and anxiety. The present study utilises a novel, network-based approach to identify (i) how well psychological processes identified in extant literature account for the relationship between pain severity and affective disorder symptoms, and (ii) which of these psychological processes are most influential for explaining the noted co-occurrence of these conditions.

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Psychological Mechanisms Linking Affective Disorder and Pain Symptoms

Chronic pain is associated with a range of pain-related beliefs that may both arise from and contribute to these pain

symptoms. Perceptions of intense, enduring pain symptoms likely shape the sufferer's beliefs about their level of perceived disability, control over their symptoms and prognosis, and pain-related self-efficacy [10–15]. For example, Demmelmaeier and colleagues [12] found in a sample of individuals with long-term chronic pain that perceived self-efficacy, level of catastrophizing, and expectations about their pain were predictive of level of perceived disability at a 12-month follow-up. It has also been shown that these pain-related beliefs are inter-related [10, 16] and, hence, may influence each other. For instance, an individual's perceived self-efficacy in dealing with her/his symptoms has been shown to prospectively predict perceived disability [17]. Similarly, perceived disability may be the basis for determining the level of control one exerts over efforts to reduce pain symptoms. For instance, Blyth and colleagues' [18] survey of adults with chronic pain showed that individuals with greater perceived disability were more likely to use passive (e.g. taking medication, using alcohol to avoid problems, etc.) than active self-management strategies (e.g. actively initiating behaviours such as planning rest and exercising to improve one's health).

French and colleagues [19] found that pain-related beliefs were negatively associated with the extent to which individuals engage in activities to reduce or self-manage pain symptoms, suggesting that these pain-related beliefs may potentially have a self-reinforcing nature by affecting the extent to which their symptoms improve, persist, or worsen over time. Over time, these negative pain-related beliefs may also engender feelings of helplessness regarding their pain symptoms, and hence confer risk for depressive and anxiety symptoms as the individual recognises the severity of their symptoms, level of impairment, and inability to improve their health [20].

Accumulated evidence also suggests that some individuals are more likely than others to report intense pain experiences, regardless of whether the pain is of neuropathic or nociceptive origin [21]. Ramirez-Maestre et al. [22] found that individuals with fear-avoidance beliefs tended to report being more hypervigilant to signs of pain, to be fearful or concerned that certain movements may aggravate pain, and have difficulty shifting away from pain-related thoughts. Other studies have found that individuals with chronic pain with fear-avoidance beliefs are also more likely to exhibit greater intensity of pain [23, 24]. Individuals with depression and anxiety show similar biases in information processing, with particular focus on negative evaluations of the self, the world, and the future (i.e. Beck's cognitive triad; [25]), catastrophising beliefs, and underestimating one's ability to cope with difficult situations [26]. Accumulated evidence suggests that these cognitive distortions may increase perceived intensity of pain symptoms and level of disability, decrease sense of control over one's pain, and reduce self-efficacy (i.e. confidence) in being able to self-manage pain symptoms [27].

This pattern of findings highlights a complex network of interrelated and potentially mutually influencing psychological factors that may perpetuate the link between affective disorder symptoms and pain symptom experience. However, the interconnectedness of these constructs makes it difficult to identify a clear direction of effects to plausibly test using a longitudinal design. This inter-connectedness of symptoms also makes it difficult to identify the psychological factors that are most influential for the relationship between affective disorder and pain symptoms.

A Network Perspective on Psychological Influences on Affective and Pain Symptoms

One intermediary step that may help to direct future efforts at longitudinal evaluation of proposed mediation pathways is network analysis. In standard implementation [28], this analytic approach derives a visual (and numeric) network of correlations among variables from a *partial* correlation matrix. A key advantage of this network approach relative to alternatives (e.g. structural equation modelling) to evaluating symptom interrelatedness is that network analysis provides summary metrics (centrality measures) for each modelled variable that help to identify which variables may be most influential in a model, and in what capacity they may exert this influence. These summary metrics break down the influence of a variable in terms of total strength of correlations with other variables (called 'strength' in network analysis parlance), as well as average length of the shortest path from one variable to all other variables in the network ('closeness') and whether the variable functions to link other constructs in the network that are not directly related ('betweenness').

By using partial correlations between modelled variables instead of bivariate correlations, the model shows any residual relationships that exist among variables after controlling for all other modelled variables. In this way, shared variance among a variety of putative predictors is removed, and it is easier to ascertain which variables may be most influential for one or more outcome variables of interest. In cases such as the present study, where modelled variables are all anticipated to correlate to some extent, evaluation of the number and strength of these partial correlations helps to identify variables that may be most influential in the network model and for which variables they may be most influential.

The Present Study

The present study augmented correlational analyses with network analysis to gain further insights into the role of specific pain beliefs in affective disorder symptoms and pain severity among individuals with persistent pain. Based on prior research, it was anticipated that bivariate correlational analyses would confirm each of these pain-specific psychological variables (perceived disability due to pain, perceived control over pain, self-

efficacy for pain self-management, and pain-related fear-avoidance beliefs) are inter-related, and also relate to pain intensity and affective disorder symptom severity (anxiety and depression). It was further anticipated that pain intensity would be related to these affective disorder symptoms.

The proposal that affective disorder symptoms act as predictors that influence pain intensity via these psychological mediators [6–9] guided predictions for the network analysis. Specifically, it was anticipated that (1) the centrality statistics from network analysis would show the affective symptoms to have the highest strength values (given greatest breadth of anticipated links to other variables), but (2) the proposed psychological mediators (perceived disability, locus of control, self-efficacy, and fear avoidance) would have the highest betweenness values, acting as a bridge between affective symptoms and pain severity.

Method

Participants and Procedure

Following [*anonymous University*] and [*anonymous pain clinic*] ethics committees' approval for this study, 743 individuals assessed at [*anonymous pain clinic*] were originally invited to participate in the research. Informed consent was obtained from all individual participants included in the study. The final sample consisted of 169 consenting individuals with chronic pain between 22 and 80 years of age ($M = 49.82$, $SD = 11.31$) who had received treatment from [*anonymous pain clinic*] between 2007 and 2014. Entry into the treatment program is based on an intake assessment conducted by a multi-disciplinary team comprising a pain specialist physician, occupational therapist, psychologist, and physiotherapist. Diagnosis was based on the presenting symptoms via subjective report in addition to physical, psychological, and functional examinations. This final sample of 169 individuals completed a battery of questionnaires (DASS-21, PSEQ, TAMPA, SOPA-R, and PDI) at admission via self-report measures. Files were accessed for consenting participants only.

Measures

Demographics Demographic information was collected from the intake assessment form. This included current age and sex of the participant, marital status, and country of birth. Pain characteristics detailing the presenting complaint and pain location, and onset date of pain (or injury) were also collected.

Affective Symptoms Depression and anxiety subscales from the 21-item Depression Anxiety Stress Scale (DASS-21; [29]) were used to evaluate level of depressive and anxiety symptoms. Items were rated on a 4-point scale (0 = 'Did not apply to me at all' to 3 = 'Applied to me very much, or most of the time'). Possible scores for each subscale range from 0 to 21, with higher scores reflect elevated symptomatology. For the depression subscale, scores of 0–4 are considered to be in the normal range of symptom experience, 5–6 mild, 7–10 moderate, 11–13 severe, and 14+ extremely severe. Scores of 0–3 reflect the normal range of anxiety symptoms, 4–5 mild, 6–7 moderate, 8–9 severe, and 10 + extremely severe. In the present study, Cronbach's alpha was .96 for both the depression and anxiety subscales.

Pain Self-Efficacy The 10-item Pain Self-Efficacy Questionnaire (PSEQ; [30]) was used to measure confidence in participants' ability to perform a range of activities while in pain, including household chores, socialising, work, and coping with pain without medication. Items were rated on a 7-point scale (0 = 'Not at all confident' to 6 = 'Completely confident'). Scores range from 0 to 60, with higher scores indicate higher levels of pain self-efficacy. Cronbach's alpha was .92 in the present study. Both the PSEQ and DASS-21 are part of the core recommended measures currently included in the electronic Persistent Pain Outcomes Centre (ePPOC) national benchmarking evaluation, for both government-funded and privatised pain management clinics in Australia (see http://ahsri.uow.edu.au/eppoc/index.html).

Fear-Avoidance Beliefs The 17-item TAMPA Scale of Kinesophobia (TSK; [31]) was used to measure fear of movement or (re)injury and consists of two subscales: 'activity avoidance' (i.e. avoiding movements connected with physical activity) and 'somatic focus' (i.e. pain is interpreted as a sign of harmful bodily processes). Items are rated on a 4-point scale (1 = 'Strongly disagree' to 4 = 'Strongly agree'). Scores range from 17 to 68, with higher scores indicate stronger perceptions of each subscale, e.g. greater fear of movement or (re)injury [32]. Alpha was .76 in the present study.

Perceived Control The 35-item Survey of Pain Attitudes, revised version (SOPA-R; [33]), was used to measure the influence of beliefs and feelings on long-term adjustment for people with chronic pain. Items were rated on a 5-point scale (0 = 'This is very untrue of me' to 4 = 'This is very true for me'). Of the seven subscales included in the SOPA-R, the current study included the 'control' subscale only; possible scores range from 0 to 4 as items are averaged, with low scores indicating low perceived control. In the current study, Cronbach's alpha was .83.

Pain Disability The Pain Disability Index (PDI) was used to measure the self-perceived impact that pain has on the ability of a person to participate in essential life activities. Participants rate the level of interference that pain has in six broad areas of functioning: family/home responsibilities, recreation, social activity, occupation, sexual behaviour, and life-

support activity. Each area of functioning is rated on an 11-point scale (0 = 'No disability' to 10 = 'Worst disability'). Scores range from 0 to 60, with higher PDI scores indicate greater disability associated with pain. Cronbach's alpha was .85 in the present study.

Pain Intensity Average pain intensity in the past week was measured on the Numerical Rating Scale (NRS; [34]), with possible scores ranging from 0 ('No pain') to 10 ('Worst pain').

Analytic Strategy

Network analyses were conducted using the *qgraph* [35], *parcor* [36], and *bootnet* [37] packages within the statistical platform R. The adaptive least absolute shrinkage and selection operator (LASSO) approach was implemented as an efficient means for eliminating spurious, non-zero correlations [38].

The generated network consists of variables (referred to as nodes in network analysis), connected by lines (edges). The width and colour of edges indicate the strength (with wider edges indicating stronger partial correlations) and direction (positive correlations typically as lines of one colour and negative correlations as lines of another colour) of correlations between nodes. The absence of an edge directly connecting two nodes may indicate that, after adjusting for all other nodes in the model, the remaining correlation between these two nodes is not reliably different from zero. It should be noted though that it is possible that the absence of an edge using the LASSO approach represents a false negative. Furthermore, even if two nodes are not directly connected via an edge, they may still be associated via a third node that links them together indirectly (see discussion of the concept of betweenness, below). The location of nodes within the network helps to identify clusters of nodes. Nodes cluster within the network when they are strongly correlated. The more peripherally a node is located, the less correlated it is to other nodes.

As with other statistical techniques, visual scrutiny does not always lead to conclusive interpretation of results. Consequently, quantitative centrality measures (strength, closeness, and betweenness) are also reported in order to help identify nodes that are most influential in the network. *Strength* refers to the overall relation of a node to others in the model, and is calculated by aggregating all (absolute values of) partial correlations involving a particular node. Higher scores reflect greater strength of connectivity within the model. *Closeness* refers to how far away a node is to other nodes within the network, and is calculated as the inverse of partial correlations between nodes. The notion of closeness is that the stronger a partial correlation between two nodes, the stronger the proposed statistical influence of one node on the other may occur. *Betweenness* refers to the extent to which a node acts in the model as a bridge between two nodes that are not directly related. More than this, as there may be many ways that two nodes may indirectly relate (e.g. through multiple bridging nodes), betweenness focuses on instances where a node is the shortest path (in terms of distance) from one node to another.

The *bootnet* package in R [37] was used to evaluate stability of centrality metrics from the present analyses. The correlation stability (CS) coefficient indicates the proportion of participants that can be dropped from the original sample while maintaining a correlation of 0.70 or above for centrality metrics between the full and subsetted sample. Epskamp et al. [37] recommend a CS coefficient of at least .25 to ensure sufficient stability in centrality metrics to interpret findings from one's sample, but note that these are provisional until more definitive guidelines are established.

Results

Sample Characteristics

The sample comprised of 98 females (58%) with a mean age of 50.85 years (SD = 11.40; Range = 24–80) and 71 males (42%) with a mean age of 48.39 years (SD = 11.12; Range = 26–71). Of the participants included in this study, 66.9% were Australian born. Fifty-six percent were married, 19.5% were single, 11.3% were separated or divorced, 4.7% were in a de-facto relationship, and 1.8% were widowed.

Chronic pain conditions varied considerably among participants: 87.6% reported general musculoskeletal pain (e.g. back, limbs), 8.3% widespread pain (e.g. fibromyalgia), 2.4% specific whiplash pain, and 1.8% headache. Among the participants in this sample, 56.2% ($n = 95$) had pain at three or more locations, 26.6% ($n = 45$) had pain at two locations, and 17.2% ($n = 29$) had pain at a single location. Fifty five percent ($n = 94$) of participants acquired chronic pain in the past 0–5 years, 21.3% ($n = 36$) in the past 5–10 years, 14.8% ($n = 25$) in the past 10+ years, and 8.3% ($n = 14$) did not specify a pain onset date.

Severity of Depression and Anxiety Symptoms

Descriptive statistics and correlations are presented in Table 1. Means and standard deviations for the depressive ($M = 10.31$; $SD = 6.46$) and anxiety ($M = 5.82$; $SD = 5.82$) symptom scales of the DASS-21 both represent moderately severe levels. On average, scores on pain intensity are consistent with an assessment of severe pain [38]. Perceived disability and control were also slightly higher than in past studies [39, 40], and both pain self-efficacy and fear-avoidance levels were consistent with prior pain samples [30, 41].

Table 1 Means, standard deviations, and inter-correlations among pain-related factors implicated in depressive and anxiety symptoms

	1	2	3	4	5	6	7
1. Depressive symptoms	1	.75**	.28**	.42**	− .48**	.62**	− .48**
2. Anxiety symptoms		1	.25**	.35**	− .40**	.49**	− .36**
3. Pain intensity			1	.37**	− .36**	.26**	− .26**
4. Perceived disability				1	− .64**	.47**	− .35**
5. Pain self-efficacy					1	− .45**	.47**
6. Fear-avoidance beliefs						1	− .45**
7. Perceived control							1
M	10.31	7.23	8.79	43.79	20.30	45.71	1.58
SD	6.46	5.82	1.14	9.56	11.02	9.32	0.77
Possible range of scores	0–21	0–21	0–10	0–60	0–60	17–68	0–4
Actual range of scores	0–21	0–21	5–10	15–60	0–55	20–68	0–3.8

** Correlation is significant at the 0.01 level

Depressive and anxiety symptoms were strongly correlated. Both variables were also significantly associated with pain-related beliefs (perceived disability, pain self-efficacy, fear avoidance, and perceived control) and pain intensity, with stronger magnitude of correlation with the proposed psychological mediators than with the proposed outcome (pain severity). There was also substantial inter-relation among the pain-related beliefs. All inter-relations were in the expected direction.

Network Analyses

Figure 1 shows the network (left-hand side) and associated centrality measures (right-hand side). The network shows that, controlling for other variables in the model, pain intensity and the affective disorder symptoms (anxiety and depression) did not share an edge. Depressive symptoms did, however, have edges with the perceived psychological mediators of the affective symptom-pain intensity

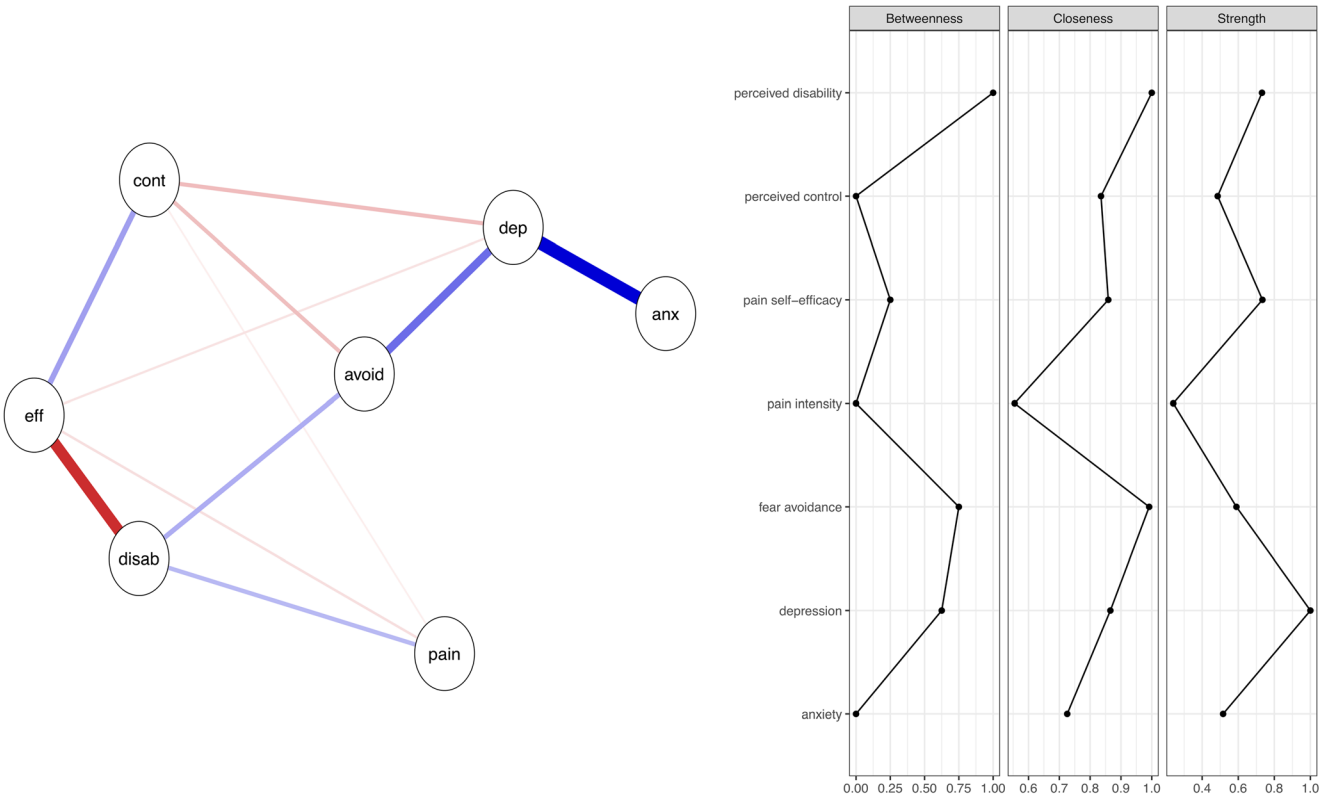

Fig. 1 Network (left-hand side) and associated plot of centrality measures (right-hand side) for modelled variables. Heavier lines (edges) between variables (nodes) in the network indicate stronger partial correlations between these variables. Red lines indicate negative partial correlations, whereas blue/purple lines reflect positive partial correlations. Anx = anxiety symptoms, avoid = fear avoidance, cont = perceived control, dep = depressive symptoms, disab = perceived disability, eff = self-efficacy, pain = pain intensity

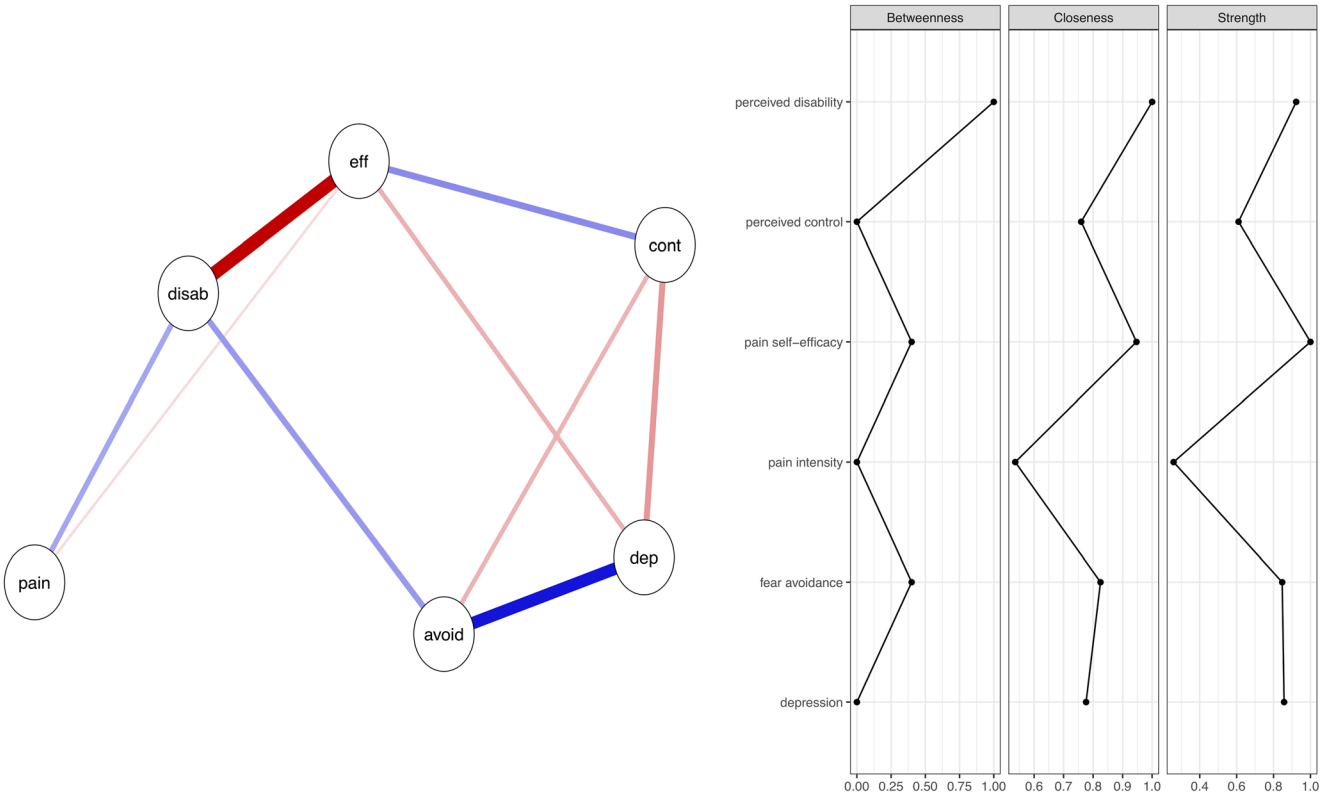

Fig. 2 Network (left-hand side) and associated plot of centrality measures (right-hand side) for modelled variables, excluding anxiety. Heavier lines (edges) between variables (nodes) in the network indicate stronger partial correlations between these variables. Red lines indicate negative partial correlations, whereas blue/purple lines reflect positive partial correlations. Avoid = fear avoidance, cont = perceived control, dep = depressive symptoms, disab = perceived disability, eff = self-efficacy, pain = pain intensity

relationship. Furthermore, there were correlations among these psychological factors: fear avoidance, perceived control, and self-efficacy, and pain intensity had edges with perceived disability and self-efficacy. With the exception of depressive symptoms, none of the modelled variables had edges with anxiety. Thus, the shortest statistical path from anxiety to pain intensity in the model was via depressive symptoms. Furthermore, the strongest edges in the model were between (1) depressive and anxiety symptoms, (2) self-efficacy and perceived disability, and (3) avoidance and depressive symptoms.

Consistent with the expectation that the modelled variables play different roles in the relationship between affective symptoms and pain severity, the centrality measures indicated that there was not a single variable in the model that was highest across all three measures (betweenness, closeness, and strength). Depressive symptoms had the highest strength value. This was due to a strong connection with anxiety, as well as having direct edges with three other nodes in the network. Surprisingly, depressive symptoms also had relatively high (although not highest) levels of betweenness, due to serving as the shortest (and only) path to anxiety for other variables in the model. Perceived disability and fear avoidance were involved in the shortest paths to other variables in the model, both as a bridging variable (highest betweenness values) and as edges with these variables (closeness values). Finally, although pain self-efficacy had a relatively low betweenness value, it had a reasonably high closeness value, due to its direct correlation with four other variables in the model.

In light of the sizable edge between depressive and anxiety symptoms, it is possible that the relative strength of the nodes over-represented the importance of depressive symptoms in the network. Accordingly, the network analysis was re-conducted after removing anxiety (Fig. 2). With anxiety symptoms excluded from the model, depressive symptoms dropped from the node (variable) with the highest strength value to third highest. Its betweenness value also dropped as it was no longer bridging other variables with anxiety. Depressive symptoms retained edges with control, avoidance, and self-efficacy, as per the original model. In this revised model, there was still no edge connecting pain intensity and depression, ruling out the possibility that the lack of direct correlation in the original model was due to heavy overlap between depression and anxiety symptoms.

Correlation stability coefficients for both the original and revised network were acceptable for strength (CS = .44 for original, CS = .52 for revised network), but suggested the need for caution in interpreting relative betweenness (CS = .05 for both networks) and closeness (CS = .13 for both networks) values among nodes.

Discussion

A variety of psychological constructs have been shown to at least partially mediate the relationship between affective disorder symptoms and experience of pain among individuals with chronic pain, including fear avoidance, beliefs about whether they are in control of their pain management (locus of control) and capable of acting to reduce their pain (self-efficacy), and perceived level of disability [8, 9, 13, 42–45]. Evidence also suggests considerable overlap between these constructs [8–10, 46–48], and potential bi-directional flow of effects among them [49, 50], complicating efforts to ascertain which of these psychological mechanisms may be most influential in explaining the link between affective symptoms and pain experience. The present study utilised an innovative analytic technique (network analysis) to gain further insights into the cross-sectional inter-relationships among these psychological variables and pain symptoms. These insights and their potential implications are detailed below.

First, although affective disorder symptoms and pain intensity were significantly related in a bivariate context—consistent with prior studies that have also found small to moderate correlations [e.g. 51–53]—there was a lack of direct correlation between affective disorder and pain symptoms in the generated network after controlling for other variables within the model. Thus, this pattern of findings is consistent with the notion that affective symptoms influence other psychological factors, such as self-efficacy, locus of control, and fear avoidance, which in turn lead to perceptions of pain intensity. Indeed, prior studies that have tested the mediating influence of psychological factors for the relationship between pain intensity and affective disorder symptoms have found evidence of full mediation (that is, the direct effect is non-significant after controlling for indirect, mediating effects) [43, 45, 54].

Second, aside from a connection between depressive and anxiety symptoms in relation to one's pain experience in the network, anxiety was not directly related to any other constructs in the psychological network of pain experience. This may be partially explained by the strong correlation found between anxiety and depressive symptoms, leaving limited variance remaining for anxiety symptoms to correlate with other variables. This might explain, for instance, why anxiety symptoms had a moderate-to-strong correlation with fear avoidance in the bivariate context yet no direct edge linked them in the network analysis. However, it should also be noted that the relationships with psychological factors and pain experience were consistently weaker for anxiety than for depressive symptoms. One possible explanation for this is the DASS-21 anxiety measure may be more heavily influenced/confounded with co-morbid medical conditions and medication side effects than the depressive symptom measure (i.e. 4 of the 7 items including dry mouth, breathing difficulty, racing heart, and trembling hands).

Third, the centrality measures suggest that self-efficacy, perceived disability, and fear avoidance may be statistically influential for both pain intensity and affective disorder symptoms, but in potentially different ways. Fear avoidance and perceived disability had high betweenness values (indicating that they were on the shortest path linking several other variables in the model) and closeness values (indicating potentially stronger partial correlations with other variables in the model). However, whereas fear avoidance was directly linked to depressive symptoms and perceived control, perceived disability directly was linked to pain intensity and self-efficacy. Their range of correlations with other modelled variables, as well as their betweenness values suggesting that they may serve as bridging variables, indicate that these may be suitable candidates for longitudinal investigation as potential risk factors for development and maintenance of affective disorder and intensity of pain symptoms. The finding that fear avoidance and perceived disability seem to link directly to different variables from each other suggests that both constructs may warrant further attention, and may be expected to behave differently in relation to affective disorder symptoms and intensity of pain symptoms.

While pain self-efficacy had a low betweenness value, it had moderately high closeness and strength values, and a diffuse range of direct links to other constructs in the network. Such a pattern suggests that, while it is not the only link to any of these modelled variables, it has some sizable direct correlations with them that remain after controlling for other noted predictors. Although the cross-sectional nature of these data preclude conclusions about causality or direction of effects, when viewed in conjunction with prior longitudinal findings of self-efficacy influencing these other variables [14, 17] and intervention studies showing self-efficacy as predictive of pain reduction [55], present findings highlight the statistical influence of self-efficacy in this network. Further research is needed to determine whether self-efficacy improvements in the course of treatment precede and moderate improvements in pain symptoms and/or other psychologically based secondary outcomes (such as fear avoidance, affective symptoms, and perceived disability).

Finally, although perceived control had direct links to other variables in the model, it had a low betweenness value, indicating that it is not the most expedient way to link one variable to another. Fear avoidance may be a stronger bridge to depressive symptoms, and perceived disability appears to be a stronger link to pain-related self-efficacy. Thus, while the present findings do not completely discount the possibility that perceived control acts as a statistical influence (direct or as a mediator) on other modelled constructs, it is clear that other proposed predictors and mediators in the model are more important. Consequently, subsequent research may prioritise testing the role of these other proposed risk factors over perceived control.

Clinically, it makes sense that fear-avoidance and depressive symptoms are directly linked. Avoidance of feared situations is likely to result in reductions in socialisation, functional capacity, enjoyable activities, and purposeful/meaningful activities and, hence, result in the development of poorer self-perception [21,

56–58]. It also makes sense that perceived disability and self-efficacy are closely linked. Having low confidence in one's ability to *manage* pain is understandably associated with feeling more *disabled* by that pain, and conversely, feeling more able to *manage* pain across various situations is associated with feeling more *able* despite the pain [21]. However, to be able to *manage* one's pain does not necessarily mean to be able to *control it*, as highlighted by the emergence of Acceptance and Commitment Therapy as an efficacious alternative to traditional cognitive behavioural therapy for a subset of individuals with persistent pain [59, 60].

Limitations

This study was cross-sectional and the positive correlations found do not permit causal inferences to be made. It is possible that the variables identified as statistically influential in the network analysis co-occur because they have a shared, generic common cause with a wide range of modelled variables. If such an explanation holds true, then the partial correlation nature of this analysis suggests that the underlying third variable is not among those modelled in the present study. Alternatively, these statistically influential variables may instead be a common outcome for these variables. Further testing, with a longitudinal network, may help to disentangle direction of effects, while maintaining the advantages of network analysis (e.g. its focus on the aggregate influence of a variable in the whole model rather than for a single outcome).

Secondly, self-report is considered the gold standard of pain measurement given its consistency with the 'subjective' definition of pain [61]. However, limitations are inherent in the subjective nature of self-reported measures, such as ability to discern and effectively communicate one's pain state. It is thus reassuring that the present sample comprises individuals with chronic pain whose symptoms were evaluated by a multi-disciplinary team involved in their treatment planning and implementation.

Thirdly, the participants in the present study comprise approximately one-quarter of those invited to the study. Although we were not provided permission to use data from the remaining invitees as a basis for comparison, it is possible that the sample used in the present study may not reflect the target population in terms of profile of symptoms. On the one hand, the higher proportion of mid- to older-aged adults, more women than men, and greater proportion of pain due to general musculoskeletal pain in the current study is consistent with prior estimates of chronic pain in the general population [62, 63] and chronic pain populations [1]. On the other hand, the high number of participants who have experienced chronic pain for longer than 5 years is likely a reflection of sampling from a pain clinic rather than the general population. These individuals also tended to have higher levels of depressive symptoms than prior work in an Australian context [64, 65]. The greater duration of pain and elevated affective symptoms in the present study may reflect greater inclination of individuals with enduring chronic pain to participate in research about their experiences.

Finally, the low sample size warrants consideration. Observed findings may be viewed as the strongest links to emerge, given the current sample size. Further, low sample size may adversely affect stability of centrality metrics. The strength metric was shown to be stable in the current sample, and hence conclusions about the relative rank of variables in terms of strength may be valid. The very low stability found for betweenness and closeness statistics suggests we should be cautious in interpreting the relative rank of variables in terms of these latter statistics.

Conclusion

Despite these limitations, the present study offers several key insights into the relationship between pain intensity and affective symptoms. While present findings confirmed the cross-sectional relationship between affective disorder symptoms and pain intensity observed in prior studies [51–53], this relationship disappeared in the network analysis due to the presence of proposed psychological mediators. Further, despite observed inter-relations among the psychological factors, self-efficacy, fear avoidance, and perceived disability were each identified as having statistical influence in the network model. Whereas self-efficacy tended to have more direct correlations with other variables, both fear avoidance and perceived disability often served as links between other variables, and may potentially act as mediators that account for the inter-relations of other symptoms in this network. Longitudinal investigation in which these variables are manipulated is needed to confirm or disconfirm predictions about the nature and direction of their influence on the other constructs in the presently tested network.

Compliance with Ethical Standards

Conflicts of Interest The authors declare that they have no conflicts of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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