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A Network Analysis of Fear-Avoidance Beliefs, Mood, and Disability in Chronic Pain

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Objectives: Although the multifactorial nature of chronic pain is well established, research has predominantly examined isolated variables or singular pathways that may contribute to this condition. We use a complex systems perspective to examine the interplay of psychological factors in the context of chronic pain.

Methods: We analyzed 2 cross-sectional data sets (N = 935 and 1366) collected at a pain clinic and rehabilitation center in Belgium from individuals primarily with musculoskeletal pain. These included self-reported data on pain-related fear and avoidance beliefs, depression and anxiety symptoms, pain intensity, and disability. We used Gaussian Graphical Models to examine conditional associations between these variables, their relative importance (having more/stronger relationships), and how they are moderated by pain-related fear and pain intensity.

Results: Our analyses revealed highly interrelated networks, with several unique, positive associations between the included factors. Depressive symptoms and pain intensity were most strongly related to pain disability. Fear-avoidance beliefs featured less prominently than previous studies looking at this concept in isolation have suggested. Apart from differences in 2 associations between samples, and 2 moderation effects of pain intensity in 1 sample, the network structure was similar across the 2 samples.

Discussion: Overall, our results show that psychological factors related to disability have intricate interrelations, highlighting the complexity of chronic pain and the need to study its many components in relation to each other. The consistency across the 2 samples provides encouraging evidence that the results reflect a stable pattern within this complex system.

Key Words: chronic pain, complex systems approach, network analysis, pain-related fear and avoidance beliefs, disability

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Chronic musculoskeletal pain is a common condition that greatly impacts well-being and quality of life,[1–3] and often results in disability.[4–6] The condition is highly complex, with many societal, social, psychological, and biological factors contributing.[7–9] Here, we focus on psychological factors, seeing their importance in understanding the development, consequences, and management of chronic pain.[10–12] According to the fear-avoidance model, negative beliefs about pain, pain-related fear, and avoidance are key for understanding pain disability.[13–18] In the current literature, pain-related fear and avoidance are most often defined as beliefs about pain indicating harm to the body, and the importance of avoiding activities that may lead to further harm, respectively. Several reviews and meta-analyses have shown that higher pain-related fear and avoidance are associated with higher pain intensity and pain disability across individuals, and that certain aspects of mood, such as symptoms of depression and anxiety, often co-occur with pain-related disability.[19–21]

Indeed, several models of chronic pain highlight the complex interactions between these psychological factors (among others) as important for understanding this condition.[8,9,15,22] However, most studies have examined these psychological components in isolation. Here, we address this limitation by adopting a complex systems perspective. This perspective proposes that a phenomenon such as chronic pain emerges from a system of elements that interact with each other in complex ways.[23–26] To understand the phenomenon, therefore, one should examine the characteristics of the system and look at how these elements interact with each other, instead of focusing on single elements or relationships. In the context of chronic pain, this would imply that considering pain-related fear, avoidance, anxiety, depression, pain intensity, and pain-related disability together to uncover their unique relationships promises a better understanding of this condition.

Three recent studies analyzed these components in a single model using network analyses and found dense networks of positive relationships among variables.[27–29]

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The data used in this study were collected for clinical use at the Multidisciplinary Pain Centre or the Department of Physical Medicine and Rehabilitation at a hospital in Genk, Belgium (Ziekenhuis Oost-Limburg). As per the agreement made with the hospital, the authors cannot share the data with third parties.

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Interestingly, all 3 identified pain-related disability and combined pain-related fear-avoidance beliefs to be among the variables with the most and strongest connections (ie, having the highest centrality), signaling their relative importance. Zhao et al[28] explored the moderating role of pain extent, defined as the number of body areas affected by pain, revealing that the strength of associations between psychological factors (anxiety and depression) and pain-related outcomes (intensity and disability) was lower when pain was more widespread. This suggests that network relationships may vary for different subgroups of individuals. Pain-related fear and pain intensity are especially interesting as moderators, as some have argued that pain-related fear is more disabling than pain itself, based on findings indicating that measures of pain-related fear provide better predictions of pain-related disability than pain intensity.[30] In contrast, others have emphasized that the role of pain intensity should not be underplayed, based on findings of robust and unique relationship between pain intensity and pain-related disability.[31] Although these early results highlight the relevance of investigating the complex interrelations of psychological variables related to chronic pain, more work is needed to assess the network structure, centrality metrics, moderation effects, and their stability.

In line with the complex systems perspective, here we use network analyses to describe cross-sectional conditional associations between pain intensity, pain disability, anxiety, depression, pain-related fear, and avoidance beliefs in 2 large samples of patients with chronic pain, so we can examine chronic pain as a complex system and look at its interacting elements. We modeled relationships between these variables and explored whether pain-related fear and pain intensity moderated the relationships within these networks. We expected strong positive relationships between pain-related fear, avoidance, pain disability, depression, anxiety, and pain intensity.[15,27–29,32] In addition, we expected pain disability and pain-related fear to be among the variables with the highest centrality,[27–29] and higher levels of pain-related fear and pain intensity to be associated with more and stronger connections in the network.

METHODS

The analyses conducted for this study were preregistered on OSF as a Secondary Data Preregistration (see https://osf.io/7nmx2/?view_only=542fd4c0c0964c79b17f47032b5f2184). We kept track of post-registration changes in a document that can be found on the associated OSF page (https://osf.io/vyg3b/?view_only=4f09cf46eccf48d9b9692f0412c7a045).

Participants

The data set consisted of self-reported survey data from persons seeking care for chronic pain at the Multidisciplinary Pain Centre or the Department of Physical Medicine and Rehabilitation at a hospital in Genk, Belgium (Ziekenhuis Oost-Limburg). Data were collected at 2 sites between May 2015 and December 2022, creating 2 samples, which we analyzed separately. Participants either came for multidisciplinary examination and diagnosis (sample A; N = 1594), usually after medical treatment did not alleviate pain sufficiently, or for a rehabilitation program consisting of 36 bi-weekly sessions geared towards alleviating functional limitations related to low back and neck pain (sample B; N = 1479). For the first group, only 1 measurement was taken before the multidisciplinary assessment, whereas for the second group, there was a pre-intervention and post-intervention measurement. In the current study, we only included pre-intervention data. Accordingly, we performed only cross-sectional analyses. Demographic and clinical information was collected from the participants' electronic files.

Questionnaires

Participants filled out the surveys at home in the days before their appointment, or in the waiting room of the hospital right before or immediately after their appointment. The questionnaires included the Tampa Scale of Kinesiophobia (TSK[33–35];), the Pain Disability Index (PDI[36,37];), the Hospital Anxiety and Depression Scale (HADS[38,39];), Multidimensional Pain Inventory (MPI[40,41];), Oswestry (Low Back Pain) Disability index (ODI[42,43];), and the Neck pain Disability Index (NDI[44,45];). Although the original 17-item version of the TSK was administered, we only used the items included in the 11-item version for our analyses, as this version has shown a better factor structure across samples with various chronic musculoskeletal pain conditions in 3 countries.[34] Not all questionnaires were administered to everyone; in sample A, participants filled out the TSK, HADS, PDI, and MPI, whereas in sample B, participants filled out the TSK, HADS, ODI, and NDI. The questionnaires were administered in Dutch and have been validated in Dutch samples.[34,37,39,41,43,44]

TABLE 1. Variable Definitions

Variable	Definition	Measurement
Pain-related fear	Self-reported beliefs about the meaning of pain in terms of current and/or future harm to the body	Sum score of items 3, 5, 6, 7, and 11 of the TSK[46]
Avoidance	Self-reported beliefs about the importance of avoiding activities to prevent further pain or harm	Sum score of items 1, 2, 10, 13, 15, and 17 of the TSK[46]
Depression	Self-reported feelings of depression (eg, [lack of] enjoyment, interest, excitement)	Sum score of even-numbered items of the HADS[47]
Anxiety	Self-reported feelings of anxiety (eg, worry, restlessness, tension, etc.)	Sum score of odd-numbered items of the HADS[47]
Pain-related disability	Self-reported interference of pain in everyday activities	Sample A: sum score of PDI[48] Sample B: sum score of NDI or ODI*[40,49]
Pain intensity	Self-reported intensity of pain at the moment of measurement	Sample A: item 1 of MPI Sample B: item 1 of NDI

*For each participant, either the ODI or NDI sum score was used, based on which was higher for that person. For both the NDI and ODI, item 1 was not included in the sum score as this item measures pain intensity.

From these questionnaires, we constructed 6 variables for our main network analyses (see Table 1 for their definitions). As participants in sample B filled out questionnaires on disability both due to neck pain (NDI) and low back pain (ODI), for each participant we selected the sum score of the disability questionnaire that was highest for them (excluding the first item in each questionnaire, as they both measure pain intensity). We chose item 1 of the NDI to measure pain intensity, rather than item 1 from the ODI, as it is more straightforward (NDI: ranging from "I have no pain" to "I have the worst imaginable pain"; ODI: ranging from "I can endure the pain without painkillers" to "Painkillers don't help therefore I don't use them"). For most constructs, the correlation among items as captured by Cronbach's alpha was within the range of values generally considered acceptable (0.74 to 0.86), whereas for pain-related fear in both samples, and avoidance in sample B, Cronbach alpha was somewhat lower (0.66 to 0.67). As many issues have been cited concerning the use of Cronbach alpha, we also calculated MacDonald's omega.[50,51] This metric yielded comparable results (see section 3 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231).

Missing Data and Sample Size

One of the surveys in our study (MPI) was no longer administered at 1 of the sites after July 2019, so patients coming after this time were not included (N = 655). Apart from this, 117 participants (4.8%) had no data for 1 or more of the questionnaires used in the project. As all or most items on at least 1 of the questionnaires were missing, these participants were also excluded from the study. Bayesian comparisons indicated that there was moderate-strong evidence ($BF_{01} < 0.33$[52];) for differences between the included and excluded samples on a subset of items (11/33 for sample A, 32/45 for sample B). The excluded sample tended to score higher on these items, with a few items showing a higher score for the included sample ($BF_{01} > 3$), although these tended to be small (difference score of 0.11 to 0.88 on a 4, 6, or 10-point scale). For the remaining items the evidence was either indicating no difference or was inconclusive (see section 1 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231).

Beyond this, few data were missing (0.1% to 2.1% for certain items of the TSK, HADS, ODI, and NDI). Overall, people who missed 1 item in a questionnaire were more likely to miss another (correlations up to 0.50), but this did not seem to be related to scores on the questionnaires in most cases (correlations < 0.10). For a few item combinations of the TSK, there were weak negative correlations between the missingness pattern and item data (−0.10 to −0.22), meaning for these items, more missing data were associated with lower values. We imputed missing data points with the mean rating of that (sub)scale within an individual. To ensure this imputation did not affect our results, we repeated the analyses with complete cases only (see section 8 of the Supplementary Materials for results, Supplemental Digital Content 1, http://links.lww.com/CJP/B231). The final sample sizes were N = 935 for sample A and N = 1366 for sample B.

Instead of the power analyses typically conducted for univariate statistical analyses, we conducted simulations to examine the performance of different network models with the available sample sizes in our data set. We based our expected weighted network on the results of Zhao et al,[28] as they included similar variables to those in our study. These simulations indicated that, with sample sizes of ~900 and ~1400 participants, we could expect low estimation errors (mean absolute error of ~0.02 for partial correlations between −1 and 1), high correlations between the estimated and true network (~0.99), and high sensitivity for the presence of nodes (0.92 to 0.94), when estimating the network model with mgm.[53] See section 2 of the Supplementary Materials for details, Supplemental Digital Content 1, http://links.lww.com/CJP/B231.

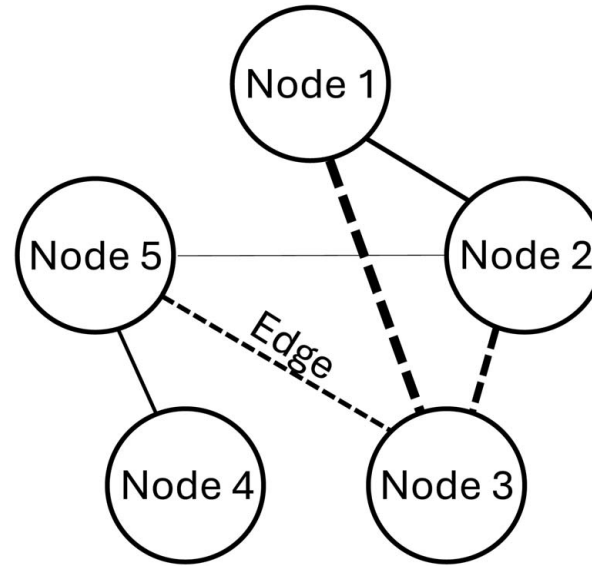

FIGURE 1. Illustration of a network model in which each variable is presented as a circle (nodes), whereas the lines between them (edges) represent the partial correlations between them. Thicker lines indicate stronger partial correlations. The dotted edges are highlighted to illustrate how node centrality is calculated.

Analyses

Model Assumptions

We explored the distribution and skewness of the data using histograms and Q-Q plots. We also looked at scatterplots of each combination of variables in the model to check for linearity. Although some skewness was present for certain variables (eg, depression and anxiety showed left-skewed distributions in sample B), no serious violations of model assumptions were identified (see section 4 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231).

Network Models

We used a Gaussian Graphical model (GGM) to model the relationships in our networks. In essence, these models estimate partial correlations between each set of variables while controlling for all others. They can then be presented in a network in which each variable is a node, whereas the edges between the nodes represent the partial correlations, with thicker lines indicating stronger partial correlations (Fig. 1). As such, GGM models highlight the unique and direct relationships that remain between variables when considering all the others. We estimated the GGMs using the mgm package using default settings.[53] This involved fitting node-wise linear regression models with

least absolute shrinkage and selection operator (LASSO) regularization using 10-fold cross-validation for parameter selection. This procedure searches for the smallest set of predictors that can accurately predict the outcome by shrinking regression coefficients towards zero and adding a penalty for more complex models. We used additional thresholding of estimates based on Loh and Wainwright[54] to control for false positives. We used the same function and estimation method to conduct our moderation analyses (moderated network model[55];). We performed 2 moderation analyses for both samples, which examined the moderation effect of pain-related fear and pain intensity on the relationships between the remaining variables in the model.

Centrality Metrics

On the basis of the estimated networks, we calculated both node strength and betweenness centrality using the centralityTable function from the bootnet package.[56] Node strength was calculated by summing the absolute partial correlations (edges) each node had with the other nodes. For example, the node strength of node 3 in Figure 1 would be calculated by adding up all the values represented by the dotted lines. As such, variables with higher node strength have more and/or stronger direct partial correlations with other variables, which is often taken as an indication of their importance within the system. Betweenness centrality represents how often the shortest path between all pairs of nodes in the network goes through each specific node. For example, node 5 in Figure 1 has high betweenness centrality as all paths between node 4 and the other nodes go through node 5. Nodes with high betweenness centrality thus signal a variable that may play a mediatory role for multiple other variables in the network.

Statistical Tests

The main inference we make was whether an edge (partial correlation) or a moderation effect was present in the network (ie, is non-zero), which was part of the network model estimation. We also compared the node strength and betweenness centrality metrics of different nodes to each other, resulting in 2 sets of 15 comparisons for each sample. We used nonparametric bootstrapping (5000 repetitions) to construct bootstrapped confidence intervals (99%) around the difference between the centrality values of 2 nodes and considered those that do not feature zero to be significantly different from each other (differenceTest function from bootnet[56];). This bootstrapping procedure also protects against the influence of outliers. Through a simulation study, Epskamp et al[56] showed that this test is somewhat biased, making it more conservative than expected by theory. It remains an open question how to correct for multiple comparisons in this context, as typical methods for correcting for multiple comparisons are generally not feasible, and specific methods have yet to be developed.[56] With the aim of limiting the chances of false positives due to multiple comparisons while also balancing the chance of false negatives, here we used a confidence interval of 99% instead of the more typical 95%. In our case, this is more lenient than a Bonferroni correction (ie, equivalent to $P < 0.01$ instead of $P < 0.003$ with Bonferroni).

Post Hoc Network Stability

We assessed the stability of the constructed networks post hoc, as suggested by Epskamp et al.[56] Specifically, we assessed the accuracy of the partial correlations and the stability of the centrality measures using bootstrapping procedures (nonparametric and case-dropping procedures, respectively, 5000 bootstraps for each). We quantified the stability of centrality measures using the CS-coefficient, calculated using the corStability function with default settings. This metric represents the maximum proportion of participants that can be dropped before the correlation with the original centrality measure falls below 0.7, with 95% certainty. In line with Epskamp et al,[56] we considered centrality measures with a CS-coefficient > 0.5 to provide a reasonably stable estimate of centrality. In addition, we examined the predictability of nodes, which provides a sense for how variables predict each other on an absolute scale (using the proportion of explained variance R^2 in our case[57];).

Comparison of Networks Across Samples

We used the ggm_compare_estimate function from the BGGM package to compare the networks estimated for samples A and B with a Bayesian approach. As we did not have a clear prior, we iterated through several values for the prior_sd parameter (from 0.25 to 0.75) to test the influence of the prior on the results (ie, a sensitivity analysis[58];). Apart from that, we used default settings.

Software and Scripts

Data curation before anonymization was conducted in Microsoft Excel (Microsoft Corporation, 2019. Microsoft Excel. Retrieved from https://office.microsoft.com/excel). After that, all work was conducted using R Statistical Software (v4.2.2[59];). The core packages we used include mgm (v1.2.13[53];), bootnet (v1.5[56];), qgraph (v1.9.3[60];), inet (v0.1.0[46];), and BGGM (v2.0.4[58];). The scripts used in this study are available through OSF (https://osf.io/vyg3b/?view_only=4f09cf46eccf48d9b9692f0412c7a045).

Positionality Statement

Positionality statements inform readers about the perspective and experience authors bring to the presented research, and have long been used in qualitative research settings (eg, see[61]). In line with increasing calls to provide such context for quantitative research as well (eg,[47,48,62]), we offer a positionality statement to highlight relevant aspects of our identities and how they relate to our study.

As a team, we share certain relevant aspects of our identities, whereas in other areas we each bring our own unique experience. We are all white and cisgender, live and work in Western Europe, and bring expertise to this project from the field of health psychology. For one of us, this comes from working with people who experience chronic pain in clinical practice, whereas for the rest this experience primarily stems from scientific work. Among us, we bring together knowledge of the scientific study and theory of psychological factors related to chronic pain, as well as expertise in research methodology. Our team is fairly well balanced in terms of gender identity and years of experience, as it consists of both women and men, as well as people in the early, middle, and later stages of their career. Some of us have experienced pain for a short duration and/or of a mild intensity; however, none of us has lived experience similar to the participants in this study, therefore, the insider perspective of living with long-term, disabling chronic pain is not represented in this paper.

TABLE 2. Demographic and Clinical Characteristics for Samples A and B (Means ± SD), and Their Statistical Comparison

	Sample A (N = 935)	Sample B (N = 1366)	Statistical comparison
Sex (F/M)	586/349	742/624	BF = 0.006, strong evidence for difference
Age	48.4 ± 11.95 (12-83)	46.0 ± 11.84 (15-81)	BF < 0.001, strong evidence for difference, Cohen's d = 0.21
Pain location	389 back; 138 neck; 52 neck & back; 88 widespread; 100 other location; 25 multiple locations; 143 not specified	1067 back; 133 neck; 108 neck & back; 5 widespread; 17 other location; 1 multiple locations; 35 not specified	NA
Employment status	256 paid work; 135 unpaid/no work; 11 in training; 229 on sick leave/ disability allowance; 45 retired; 259 not specified	770 paid work; 156 unpaid/no work; 30 in training; 199 on sick leave/ disability allowance; 84 retired; 127 not specified	NA
Marital status	64 cohabiting; 104 divorced; 534 married; 188 single; 28 other; 17 unknown	191 cohabiting; 80 divorced; 753 married; 296 single; 41 other; 5 unknown	NA
Nationality	824 Belgian; 55 Dutch; 22 other European; 9 other non-European; 25 unknown	1157 Belgian; 20 Belgian + other; 72 Dutch; 52 other European; 25 other non-European; 40 unknown	NA
Pain-related fear (TSK)	12.78 ± 2.99	12.25 ± 2.97	BF = 0.003, strong evidence for difference, Cohen's d = 0.18
Avoidance (TSK)	20.07 ± 4.88	19.08 ± 4.11	BF < 0.001, strong evidence for difference, Cohen's d = 0.22
Depression (HADS)	8.75 ± 4.53	6.79 ± 4.16	BF < 0.001, strong evidence for difference, Cohen's d = 0.45
Anxiety (HADS)	9.63 ± 4.45	8.20 ± 4.47	BF < 0.001, strong evidence for difference, Cohen's d = 0.32
Pain disability (PDI/NDI/ODI)*	0.55 ± 0.21	0.42 ± 0.20	BF < 0.001, strong evidence for difference, Cohen's d = 0.65
Pain intensity (MPI/NDI item)*	0.68 ± 0.22	0.35 ± 0.21	BF < 0.001, strong evidence for difference, Cohen's d = 1.58

*As pain disability and pain intensity were measured using different scales, these variables were scaled from 0 to 1 before comparison.

F/M indicates female/male; HADS, Hospital Anxiety and Depression Scale; NDI, Neck pain Disability Index; MPI, Multidimensional Pain Inventory; ODI, Oswestry (Low Back Pain) Disability index; PDI, Pain Disability Index; TSK, Tampa Scale of Kinesiophobia.

RESULTS

Participants

An overview of demographic and clinical characteristics for both samples is presented in Table 2. Bayesian comparisons indicated that there was strong evidence for differences between the 2 samples on all variables for which statistical comparisons were conducted, although the differences varied in size. For example, there was a difference in pain-related fear ($BF_{01} < 0.001$) between sample A (M = 12.78, SD = 2.99) and sample B (M = 12.25, SD = 2.97), but the size of this difference was small (~0.5 on a 0 to 20 scale, Cohen's d = 0.18). Other differences were large, such as the higher Pain Intensity in sample A (M = 0.68, SD = 0.22) compared with sample B (M = 0.35, SD = 0.21; $BF_{01} < 0.001$), which displayed a difference of 0.33 on a 0 to 1 scale (Cohen's d = 1.58).

Accuracy and Stability Checks

The results from the bootstrapping procedures used to examine the accuracy of edges (partial correlations) and stability of centrality metrics are reported in detail in section 5 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231. The bootstrapped 95% quantiles around the edges were ~0.1 to 0.15 in width. This means that, in 95% of bootstrapped samples, the partial correlations between 2 specific variables fell within 0.1 to 0.15 distance of the partial correlation estimated in the main analysis, which is comparable to previously published studies using large samples.[1,63] The CS-coefficient indicated that the centrality metrics were stable for strength in both samples (CS > 0.75), and for betweenness in sample B (CS = 0.52), but not in sample A (CS = 0). Therefore, we did not examine betweenness centrality further for sample A.

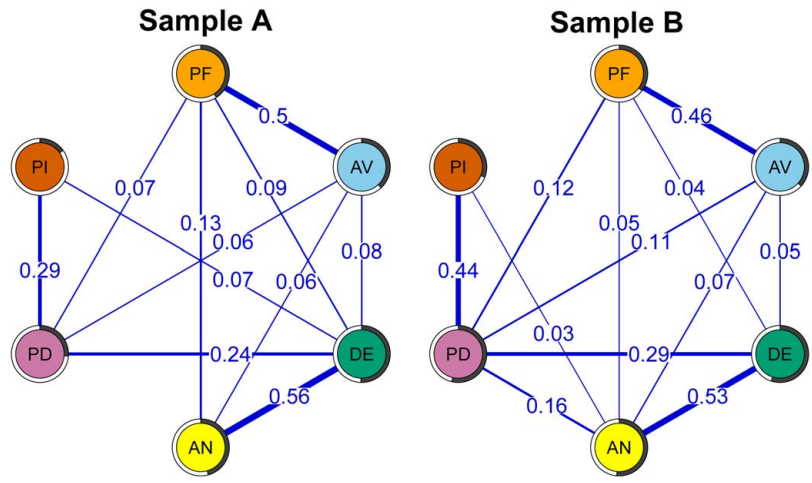

FIGURE 2. Cross-sectional network structure for sample A and sample B. The graphs were created with qgraph while ensuring that the visualizations are comparable across groups. The lines between variables (edges) represent partial correlations, with thicker lines indicating stronger partial correlations. The pie charts around each node represent the proportion of variance explained by its neighbors (R^2). AN indicates anxiety; AV, avoidance; DE, depression; PD, pain disability; PF, pain-related fear; PI, pain intensity.

Network Structure

The networks estimated from the data of samples A and B using GGMs are illustrated in Figure 2, with edges representing partial correlation coefficients. For sample A, 11 out of 15 possible edges were estimated to be present (ie, significant according to the network model estimation), and these partial correlations ranged from 0.06 (Avoidance – Anxiety) to 0.56 (Depression – Anxiety). For sample B, 12 out of 15 possible edges were estimated to be present, and these partial correlations ranged from 0.03 (Anxiety – Pain Intensity) to 0.53 (Depression – Anxiety). All edges in both networks were positive (see section 6 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231). We used qgraph to create the network illustrations, using settings that ensure that network visualizations are comparable across groups. The results of the analyses with complete cases only were highly similar (see section 8 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231).

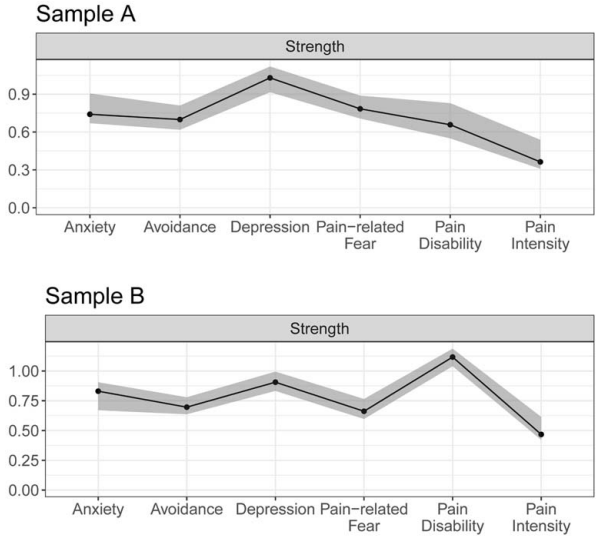

FIGURE 3. Raw centrality values for strength centrality in sample A, and in sample B. The shaded areas represent the bootstrapped 99% quantiles. AN indicates anxiety; AV, avoidance; DE, depression; PD, pain disability; PF, pain-related fear; PI, pain intensity.

Centrality Metrics

Figure 3 illustrates the raw strength centrality metrics derived from the networks estimated for sample A and sample B (betweenness centrality is illustrated in section 7 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231). Bootstrapped difference tests indicated that, for strength centrality in the network of sample A, Depression had a significantly higher centrality compared all other nodes (bootstrapped 99% quantiles did not include zero), whereas pain intensity had a significantly lower centrality than all other nodes. In the network of sample B, pain disability had a higher strength centrality than all other nodes, and a higher betweenness centrality than pain-related fear, anxiety, and pain intensity. Depression showed higher strength centrality compared with pain-related fear, avoidance, and pain intensity, whereas pain intensity had a lower strength centrality compared with all other nodes. Note that no corrections for multiple comparisons were applied (see Methods—Analyses—Statistical Tests). The raw centrality scores and bootstrapped 99% CIs for the difference tests can be found in section 9 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231.

Node Predictability

The predictability of each node, quantified as the proportion of explained variance by its neighbors (R^2), is visualized as a pie chart around each node in Figure 2. For sample A, predictability ranged from 0.15 (pain intensity) to 0.51 (depression). For sample B, predictability ranged from 0.32 (pain intensity) to 0.57 (depression). The raw predictability scores for all nodes can be found in section 10 of the Supplementary Materials, Supplemental Digital Content 1, http://links.lww.com/CJP/B231.

Network Comparison

We compared the networks estimated for sample A and sample B using a Bayesian comparison. For most edges, the test indicated moderate-strong evidence for the hypothesis that there are no differences between the 2 samples ($BF_{01} > 3$), regardless of which prior was used. For 2 edges (disability – pain intensity and anxiety – disability), there was moderate-strong evidence for a difference between the 2 samples ($BF_{01} < 0.04$), again regardless of the prior we specified. There was a stronger partial correlation between these edges in sample B compared with sample A. For 2 edges, however, the results shifted somewhat depending on the specified prior. Namely, for pain-related fear – anxiety and depression – disability, the evidence in favor of no difference between groups shifted from moderate ($BF_{01} = 5.50$ and 10.57, respectively) to anecdotal ($BF_{01} = 1.33$ and 2.54, respectively) with decreasing priors (prior SD = 0.25 to 0.65).

Moderation Analyses

The moderation analyses identified 2 moderation effects for Pain Intensity in sample A (Fig. 4). Namely, with higher pain intensity, the positive relationship between avoidance and pain-related disability became weaker (partial $r = -0.046$ per 1-point increase in pain intensity), whereas the relationship between avoidance and anxiety went from a small negative to small positive effect (partial $r = +.052$ per 1-point increase in pain intensity). Notably, however, the stability of these effects as determined with bootstrap resampling was only moderate (effects were present in 51% and 44% of bootstrap samples, respectively). Our analyses identified no other moderation effects of pain-related fear and pain intensity in sample A. No moderation effects were found in sample B.

DISCUSSION

We examined the unique relationships between pain-related fear, avoidance, depression, anxiety, pain intensity, and pain-related disability using network analyses in 2 large cross-sectional data sets. In both samples, we observed a dense network of positive relationships among variables, where pain disability and depression had especially many and strong connections to other variables (ie, high strength centrality). Apart from 2 connections, the 2 samples displayed the same network structures. Our moderation analysis revealed that the relationship between avoidance and anxiety was slightly negative when pain intensity was low, and slightly positive when pain intensity was high. In addition, the relationship between avoidance and disability was weaker for those with higher pain intensity. These moderation effects were only found with moderate stability in sample A, but not for sample B.

The most prominent features of the networks, strong positive relationships between pain-related fear and avoidance, depression and anxiety, pain intensity and pain-related disability, and depression and pain-related disability, were present in both networks and largely align with the findings of previous network studies of chronic pain.[27–29,64] These studies also observed that pain-related disability and depression were among the most central (ie, with the

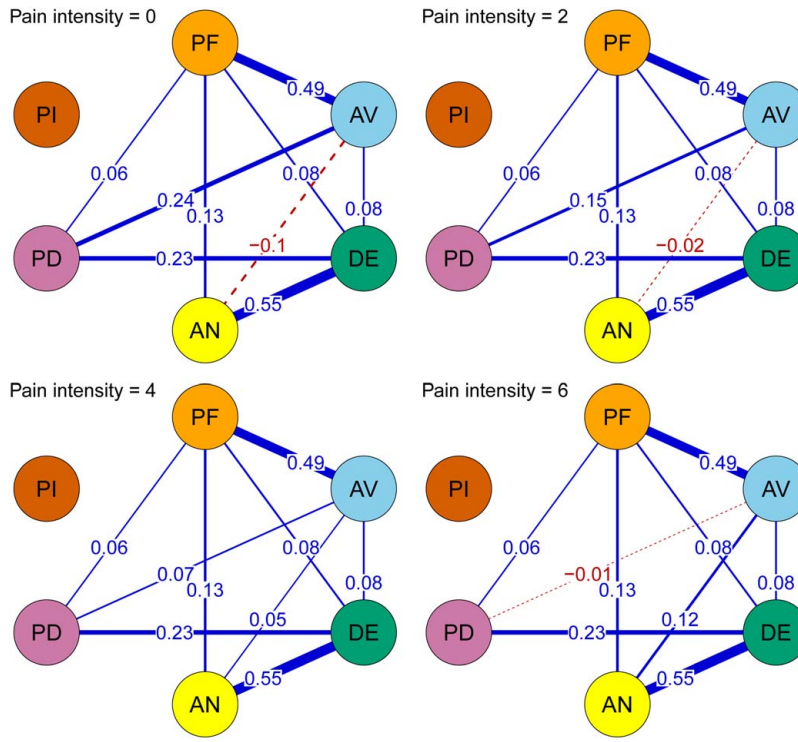

FIGURE 4. Series of network structures highlighting the linear moderation effect of pain intensity on the relationships between avoidance and pain disability, and between avoidance and anxiety in sample A. Namely, these relationships were weaker and turned from slightly negative to slightly positive, respectively, for subgroups with higher Pain Intensity ratings. The graphs were created with qgraph while ensuring that the visualizations are comparable across groups. AN indicates anxiety; AV, avoidance; DE, depression; PD, pain disability; PF, pain-related fear; PI, pain intensity.

most/strongest connections) variables. In clinical practice, pain-related disability is often the focus of treatment, as this interference with everyday activities and shrinking of an individual's world is highly burdensome to individuals experiencing chronic pain.[2,3] Our results indicate that pain-related disability has unique relationships with multiple psychological factors, even when examined together. That is, across individuals, pain-related fear and avoidance beliefs, anxiety and depression symptoms, and pain intensity all explained unique portions of variance in pain-related disability. This fits well within the picture of chronic pain as a complex condition in which multiple different factors may play a role, and further emphasizes the need for multidisciplinary management that takes into account these different factors together.[8,9]

Although pain-related fear-avoidance beliefs had a direct relationship with pain-related disability, they took up a less prominent place than expected. For example, a meta-analysis examining this relationship found a robust, moderate-strong correlation between pain-related fear and pain-related disability across studies (average sample-size weighted $r = 0.42$[65];). Of course, they considered this relationship in isolation, whereas we also incorporated and conditioned on additional psychological variables known to relate to pain-related disability. Two network studies, which did take a broader perspective, also identified moderate partial correlations and/or found a combined measure of fear-avoidance to be among the most central features of the network; however, these had relatively small sample sizes ($N = 47$ and 169[27,66];). Our findings are more in line with 2 network studies utilizing larger data sets ($N = 2241$ and 10,436; from the same registry), which demonstrated rather low centrality for fear-avoidance and small partial correlations between fear-avoidance and pain-related disability.[28,29] Overall, these findings indicate that, in contrast to earlier studies highlighting a moderate-to-strong relationship between fear-avoidance beliefs and pain-related disability, the unique relationship between these factors may be relatively small when considered in the context of other psychological factors.

Instead, depression and pain intensity had the strongest unique relationships with pain-related disability. Depression and chronic pain are closely intertwined, and having both simultaneously has been associated with worse functional outcomes.[20,21] This may be especially relevant in samples with longer pain duration.[67] Although pain intensity has been regarded as less important for understanding disability than pain-related fear and avoidance,[14,30] many studies have shown a robust relationship between pain intensity and disability.[13,31,49] Here, pain intensity had the strongest unique relationship with pain-related disability, while accounting for pain-related fear and avoidance beliefs as well as anxiety and depression symptoms. Of course, due to the cross-sectional nature of this study, we cannot establish which one causes the other or whether they cause each other. Nonetheless, this study supports the idea that pain intensity is an important factor to consider when talking about pain disability. Future studies could look into the interplay between depression, pain intensity, and other factors during intervention to see if they play a similarly prominent role in treatment success, and should thus receive special attention.

Our study also identified 2 moderation effects of pain intensity. First, the small negative relationship between avoidance and anxiety turned into a small positive relationship going from low to high pain intensity. This aligns with the idea that higher pain intensity usually has a higher threat value,[68] and thus anxiety and beliefs about the importance of avoiding activities may become more relevant at higher levels of pain intensity. Previous studies have generally found that stronger avoidance beliefs were associated with worse pain-related disability.[69–72] Our finding that this relationship was weaker for subgroups with higher pain intensity suggests that this previously observed effect may hold for populations with lower pain intensity, but not necessarily be relevant for populations with high pain intensity. That being said, bootstrapping indicated these effects were only present in about half of the random samples, so they may not be particularly stable and need further investigation before being interpreted in more depth. If such effects are consistently found in future research, this may provide indications for which factors to emphasize more/less during treatment for specific subgroups of patients.

Whereas the 2 samples generally had a similar network structure, 2 edges did show differences. As both involved pain-related disability, these differences may relate to the different measures of pain-related disability for the 2 samples. Notably, the pain-related disability items in sample A focus on how much participants are limited by their pain, whereas those used in sample B, in part, ask about how much they can do without pain increasing. Alternatively, they may stem from intrinsic differences between the 2 samples. For example, the sample from the multidisciplinary examination had higher values for all network variables, most notably for pain intensity. Of course, there may also have been differences in aspects that we did not measure. For instance, as thorough multidisciplinary examination is a relatively late step in pain treatment, sample A may have had more complex pain, and a longer pain duration compared with the rehabilitation sample. Overall, however, the relationships in the network models were surprisingly consistent across the 2 samples.

The variables were selected based on the theoretical fear-avoidance model, which describes a longitudinal process through which negative pain appraisal, pain-related fear, and avoidance behavior may contribute to the development and maintenance of pain.[14,15,17] As we used cross-sectional data, our results can neither directly support or reject the theory. Theory building is important for accumulatively constructing our scientific knowledge base, however, this has proven difficult.[73] One main challenge is the identification of robust phenomena; patterns in empirical data that can be reliably observed independent of the exact method of measurement. Once such phenomena have been established, they can be used to develop, constrain, and test theories (eg, through formalizing theories[74]), as any theory will have to be consistent with the observed phenomena.[73]. The results presented here may contribute towards establishing a robust cross-sectional network structure for the role of fear-avoidance in chronic pain. In addition, future studies could expand upon this work by taking into account additional factors, including other psychological variables as well as biological and social aspects of chronic pain, to build towards a more comprehensive biopsychosocial model. Another interesting direction would be to examine the interactions of the factors included here within individuals over time, as knowledge of such relationships could be translated to clinical practice more directly. Indeed, there is already work exploring whether individual networks based on longitudinal data can be used to optimize treatment for chronic pain.[63]

Our study has several limitations. As we used an existing data set, we were dependent on the information that was available to us. Next to biological and socioeconomic factors,[7–9] several psychological variables that are of potential interest, such as pain appraisal, resilience, and motivational factors,[13,14,18,31,75] could not be examined. Similarly, due to limited demographic and clinical data, it was difficult to describe our samples in detail and understand where differences between them may stem from. In addition, the questionnaires that were used may not have been optimal due to known issues with construct definitions and overlap.[76,77] The use of different disability questionnaires complicated the direct comparison across samples. Similarly, pain intensity was measured using 2 different scales, and seeing the multiple dimensions of pain experiences and the challenges in measuring them,[78] the use of a single item provided only a limited measure of this construct. The application of network models in psychology is fairly recent, and especially the interpretation, stability, and unique value of centrality metrics in this context is not yet clear.[79] Furthermore, this study examined associations across people at one point in time, and thus our results cannot be translated to causal and/or within-individual effects without further examination. As these data were collected at a specialized health care facility, the sample may differ from the broader population of people experiencing chronic pain in clinical (eg, severity, duration) and demographic (socioeconomic status, ethnicity) characteristics. We had little information on the cultural and immigration background of the participants in this study. However, the region where data were collected has seen a strong influx of migrant workers in the mining industry, therefore, our samples may be expected to include some diversity in backgrounds. Still, this study was conducted within a specific region and setting, which limits the generalizability of our findings to other regions of the world.

Although the complex nature of chronic pain is widely recognized, this is rarely reflected in research methodology. Our study used methods that can capture and thus start mapping this condition as a complex system. Our results indicate that psychological factors form a dense network of interacting components in the context of chronic pain. Pain-related disability had unique relationships with all other variables, among which pain intensity and depressive symptoms were the most prominent. Interestingly, when considering multiple psychological factors together, fear-avoidance beliefs were less prominent than previous studies found when looking at them in isolation. This illustrates both the complexity of chronic pain, as well as the importance of using models that can examine the interplay and relative importance of different factors. The similarities across the 2 samples and with other large sample studies suggest that our results contribute to describing a robust phenomenon in chronic pain. This, in turn, may support the development of theories to better understand and treat chronic pain in the long term.

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