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The Relationship Between Posttraumatic Stress Disorder and Chronic Pain in People Seeking Treatment for Chronic Pain: The Mediating Role of Psychological Flexibility

Psychological Flexibility, PTSD and Chronic Pain

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Abstract

Objectives: The symptoms of Posttraumatic Stress Disorder (PTSD) and chronic pain are thought to interact to increase the severity and impact of both conditions, but the mechanisms by which they interact remain unclear. This study examines the relationship between PTSD and chronic pain and whether indices of psychological flexibility mediate the relationship between these two conditions.

Methods: Standardized self-report measures of PTSD, pain severity, pain interference, depression, and psychological flexibility (pain-related acceptance, committed action, cognitive fusion, and values-based action) were obtained from 315 people seeking treatment for chronic pain who also reported at least one traumatic experience.

Results: People seeking treatment for chronic pain reporting symptoms consistent with a current diagnosis of PTSD had significantly higher levels of pain severity, pain interference, depression, and cognitive fusion and lower levels of pain-related acceptance and committed action than those reporting symptoms below diagnostic threshold for PTSD. Pain-related acceptance, committed action, cognitive fusion and depression mediated the relationship between PTSD and pain severity/interference, with pain-related acceptance being the strongest mediator from the psychological flexibility model.

Discussion: Processes from the psychological flexibility model were identified as mediators of the relationship between PTSD and chronic pain in people seeking treatment for chronic pain. The psychological flexibility model may be useful as an overarching model to help understand the relationship between PTSD and chronic pain. It is possible that targeting pain-related acceptance, committed action, and cognitive fusion (among other processes) in the treatment of chronic pain may produce corresponding improvements in comorbid symptoms of PTSD when these are present and may reduce impacts of PTSD on outcomes of chronic pain. Conversely, targeting of these processes in the treatment of PTSD may produce similar improvements for symptoms of chronic pain. Further research to evaluate these possibilities is needed.

Key words: Chronic pain, posttraumatic stress disorder, psychological flexibility, mediation.

Introduction

Individuals with chronic pain are known to be at increased risk of psychiatric disorders, particularly those involving low mood and anxiety [1-3] and Posttraumatic Stress Disorder (PTSD) [4, 5]. Evidence shows that chronic pain sufferers with comorbid anxiety, depression, and PTSD present with greater levels of pain-related impairment, respond less well to pain focused treatment, and incur greater health costs [5-11]. However the mechanisms by which psychiatric disorders and chronic pain conditions interact and exert their impacts on functioning remain unclear.

Different models have been offered that identify factors that may predispose a person to develop both PTSD and chronic pain (e.g., anxiety sensitivity) or exacerbate and mutually maintain symptoms of PTSD and chronic pain (e.g., avoidant coping style) [12, 13]. To fully assess how PTSD and chronic pain symptoms interact longitudinal studies are needed, but only a handful of such studies have been carried out to date. Jenewein, Wittman et al. (2009) administered measures of PTSD and pain intensity to 323 injured accident victims five days after hospital admission and again at 6- and 12-months later and tested the influence of pain on PTSD and vice versa. The model which best fit the data found that in the first six months after the trauma, pain intensity and PTSD symptoms were mutually maintaining, but in the second six months PTSD symptoms impacted significantly on pain intensity but not the reverse [14]. More recently, Andersen, Karstoft et al. (2016) assessed 198 individuals for pain intensity and psychological distress within 3 weeks after whiplash injury and again 3- and 6-months later. Pain catastrophizing and fear avoidance beliefs were found to mediate the effect of PTSD symptoms on pain [15].

When trying to understand the mechanisms by which PTSD and chronic pain help to maintain each other findings from cross-sectional studies can be informative, even though the temporal precedence of trauma/PTSD versus chronic pain is often unclear and firm conclusions about

the direction of causation cannot be drawn [16, 17]. There is a growing body of evidence from such studies where elevated levels of depression, anxiety, maladaptive coping, alcohol use, and negative trauma-related cognitions have been identified as mediators of the relationship between PTSD and chronic pain [18-21]. Still, clear theoretically-based, psychological dimensions that might underline these variables remain to be identified [14]. The application of a more integrative psychological model to the relationship between these two conditions might further elucidate mediating variables and could in turn lead to improved treatments.

Acceptance Commitment Therapy (ACT) is a form of psychological treatment with clear research support for the treatment of chronic pain [22]. ACT has a general aim (across all problem areas or disorders) to increase *psychological flexibility*, or the ability to contact the present moment more fully as a conscious human being and to change or persist in behavior when doing so serves successful healthy functioning [23]. Psychological flexibility is a term used to capture a combination of avoidance reducing, attention-focusing, goal-directed, and engagement-oriented behavior capacities or processes. A total of six interactive processes are specified in the model: (1) *acceptance:* the active embrace of unwanted private events (physical sensations, thoughts, images, emotions) and an alternative to experiential avoidance; (2) *cognitive defusion:* the ability to distance oneself from thoughts and not be dominated by the literal meaning of them; (3) *contact with the present moment:* ongoing nonjudgmental contact with events as they occur; (4) *self as context:* the ability to separate processes of experiencing events, especially thoughts and feelings, from the content of these events; (5) *values:* desired qualities of purposive action; and (6) *committed action:* the development of larger and larger patterns of persistent effective action linked to chosen values (rather than the avoidance/suppression of negative emotional states) [23].

There is a growing body of evidence that core processes from the psychological flexibility model are correlated with the severity of pain and pain-related impairments and that changes

in these processes mediate outcome in pain-focused treatments (ACT or otherwise) [24-27]. Likewise, there is evidence that processes from (or similar to) those described in the psychological flexibility model are correlated with the severity of PTSD and may influence treatment outcome [28-31]. There is also a study showing that a treatment targeting acceptance, cognitive defusion, committed action, and values in people with chronic pain arising from whiplash injuries resulted in improvements in pain-related disability and PTSD [32]. Other studies have examined pain-related acceptance as a protective psychological variable in trauma-exposed people with chronic pain [6, 33, 34]. However, no studies that we are aware of have investigated whether processes from the psychological flexibility model mediate the relationship between PTSD and chronic pain.

Given the available evidence, it seems plausible that processes from the psychological flexibility model may help us integrate current knowledge and better understand how PTSD and chronic pain interact to increase the severity of both conditions [35]. For example, individuals with PTSD experience detailed and highly upsetting recollections of the trauma, which elicit efforts to suppress or distract themselves from the physical sensations, thoughts/images, and emotions that accompany the recollections. Unfortunately, these efforts typically have the unintended effect of increasing the frequency of traumatic recollections and related experiences, which in turn coordinate further vigilance and avoidance. What was initially an understandable effort to avoid or suppress highly intrusive recollections of a traumatic event may lead to a wider pattern of experiential avoidance (i.e., efforts to avoid or suppress all unpleasant physical sensations, images/thoughts, and emotions), cognitive fusion (i.e., an increased focus on and adherence to the content of one's thoughts and emotions rather than what is experienced through all five senses), and a diminished engagement in a broad range of available activities that are consistent with the individual's core values (e.g., work, study, engagement with family, and friends) in favor of activities that have as their primary

aim the avoidance of negative emotional experiences. In this way, the traumatic event could be seen as creating a context of generalized avoidance, hyper focus on threat, and disengagement from healthy functioning, which in the psychological flexibility model would be described as cognitive fusion, experiential avoidance, and reduced values-based and committed action. We argue that these features that constitute *psychological inflexibility* and which may arise from either PTSD or chronic pain undermine the individual's capacity to successfully confront chronic pain or traumatic reminders, and hence increase the impact of both.

The aim of the present study is to examine the relationship between PTSD and chronic pain and in particular whether various indices of psychological flexibility mediate the relationship between these two conditions. First, we compare traumatically-exposed adults with and without PTSD on measures of psychological flexibility, pain severity, pain interference, and depression in a sample seeking treatment for chronic pain. In line with earlier research [6, 18], we expect those with PTSD to show greater pain severity, pain interference, and depression as well as lower psychological flexibility, as indexed by higher cognitive fusion and lower pain-related acceptance, values-based action, and committed action, than those without. Second, we examine whether psychological flexibility, as indexed by pain-related acceptance, values-based action, committed action, and cognitive fusion, mediates the relationship between PTSD and chronic pain, as indexed by pain severity and pain interference, at the time of referral for assessment of chronic pain. As mentioned above, mediation is best tested in longitudinal studies. However, and as reviewed above, cross-sectional studies can help identify mechanisms that explain the relationship between PTSD and chronic pain, which can then be examined in longitudinal studies. Again in line with earlier cross-sectional and treatment studies connecting psychological flexibility to PTSD and/or chronic pain [6, 24-27, 29, 31, 33, 34, 36], we anticipate that psychological flexibility will demonstrate significant

and unique mediating effects on this relationship at the time of referral, after controlling for depression, which has been found to mediate the relationship between PTSD and chronic pain in previous cross-sectional studies [18, 19, 21].

Material and methods

Participants and Procedure

Participants were 315 adults with chronic pain and traumatic exposure referred for assessment at the Pain Rehabilitation Unit at Skåne University Hospital between October 2013 and January 2015. This sample has been described in a previous study and included 71.8 % of consecutive referrals, those who reported experiencing at least one previous trauma(s) [5]. The unit is a government supported, regional specialist center providing services for chronic pain and related disability. Just prior to their first assessment at the unit, potential participants were sent a letter informing them about the study that included the measures used here, and asked them to sign a consent form and return this with the completed measures to the clinic before their first assessment. The study was approved by the Regional Ethical Review Board in Lund, Sweden (2013/381).

Measures

Multidimensional Pain Inventory (MPI): The MPI (version 2) consists of 61 items divided into three parts and each item is rated on a 7-point scale (*0 =never; 6 = very often*) [37]. Only Part 1, which asks about the perception of pain and pain-related consequences, was included in this study. The subscales pain interference, 11 items, and pain severity, 3 items, were used and the mean score was calculated for each scale. The MPI has satisfactory psychometric properties (α = .72-.90) [38] and a Swedish version was used [39].

The Posttraumatic Diagnostic Scale (PDS): The PDS is a self-report measure of PTSD symptoms related to a single identified traumatic event [40]. The PDS is comprised of 49

items broken down into four parts. Part 1 is a trauma checklist. In Part 2 respondents describe their most upsetting traumatic event. They are asked to describe when it happened, if anyone was injured, perceived life threat, and whether the event resulted in helplessness or terror. Part 3 measures the severity of the 17 symptoms listed under DSM-IV for PTSD and Part 4 measures interference from these symptoms. A total severity score (ranging from 0 to 51) based on the symptom reports in Part 3 is calculated (1-10 = mild; 11-20 = moderate; 21-35 = moderate to severe; >36 = severe). The original scale (α = .92) and the Swedish version (α = .92) used in this study possess satisfactory internal consistency and validity [5, 40]

PTSD Diagnostic Status: To assess the role of PTSD diagnostic status, as opposed to PTSD symptom severity, a group-level variable was created based on the participants' responses from the PDS. Those who self-reported a trauma, the requisite number and type of symptoms, and impairment consistent with the DSM-IV PTSD criteria were categorized as having a current diagnosis of PTSD. Those who reported a trauma but either too few symptoms or insufficient impairment were categorized as not having PTSD. To be categorized as having a current diagnosis of PTSD the individual had to meet the following criteria: (A) having experienced, witnessed or been confronted with an event that involved actual or threatened death or serious injury or a threat to the physical integrity of self or others and responded with intense fear, helplessness, or horror; (B) one or more symptoms of re-experiencing; (C) three or more symptoms of avoidance and numbing; (D) two or more symptoms of hyperarousal; (E) symptom duration of more than a month; (F) clinically significant distress or impairment in two or more areas of functioning.

Committed Action Questionnaire (CAQ-18): The CAQ measures goal-directed, flexible persistence and is comprised of 18 items, each rated on a 7-point scale (*0 = never true, 6 = always true)* [41]. The CAQ has two components consisting of positively and negatively phrased items respectively. Ratings for all items are summed (negatively phrased item ratings

are first reversed) to arrive at a total score (range = 0-108), where higher scores represent greater levels of committed action. The original English version (α =.91) and the Swedish version (α = .89) used in this study have satisfactory internal consistency and validity [41, 42].

Chronic Pain Acceptance Questionnaire (CPAQ): The CPAQ measures pain-related acceptance and consists of 20-items, each rated on a 7-point scale (*0 = never true; 6 = always true)* [43]. It includes activity engagement and pain willingness with higher scores reflecting greater acceptance of pain. The English original of the CPAQ (α = .74), and the Swedish version (α = .91) used have good internal reliability and validity [43-45].

Chronic Pain Values Inventory (CPVI): The CPVI is a 12-item measure designed to assess engagement in valued activity in chronic pain patients [46]. The CPVI first asks the respondent to rate the importance of activities in six different domains: family, intimate/close interpersonal relations, friends, work, health, and personal growth/learning, on a 6-point scale (0 = *not at all important;* 5 = *extremely important)*. They are then asked to rate the degree of success in behaving in line with their values in these six domains on a 6-point scale (0 = *not at all successful;* 5 = *extremely successful)*. Two subscale scores can be calculated: a mean success rating, which is the average of the six success ratings, and a mean discrepancy rating, which is the mean of the differences between importance and success. The mean success rating was used in this study to measure values-based action [47]. Higher scores indicate greater success. Like the English original (α = .82 for both subscales), the Swedish version (α = .84 for both subscales) used has satisfactory psychometric properties [46, 47].

The Psychological Inflexibility in Pain Scale (PIPS): The PIPS is a 12-item self-report measure designed to assess psychological inflexibility through avoidance of pain (8 items) and fusion with pain thoughts (4 items) [48]. Each item is rated on a 7-point scale (1=*never*

true; 7= *always true*), where higher scores indicate greater levels of psychological inflexibility. Only the subscale focusing on cognitive fusion was used in this study. The original Swedish version used in this study has demonstrated satisfactory reliability (α = .89 (avoidance), .66 (fusion) and .87 (total scale)) and validity [48].

Hospital Anxiety and Depression Scale (HADS): The HADS is comprised of 14-items that assess symptoms of anxiety and depression amongst patients in a medical setting. Only the depression subscale was used in this study and it contains seven items rated on a 4-point scale (0 to 3), with higher scores indicating greater severity. Consistent with the original [49], the Swedish version used in this study has demonstrated satisfactory validity and internal reliability for the total (α = .90), anxiety (α = .84), and depression scales (α = .82) [50].

Data Analyses

Participants with and without PTSD were compared on demographic variables, pain interference, pain severity, and the potential mediators (pain-related acceptance, committed action, values-based action, cognitive fusion, and depression) using independent sample t-tests. Effect sizes (Cohen's *d*) were calculated for pain severity and interference and the potential mediators with adjustments made for differing sample sizes. Effect sizes were interpreted as small (*d* = .2), medium (*d* = .5), and large (*d* = .8) [51]. Alpha levels were not adjusted for the number of analyses as it was deemed most important to avoid making Type II errors given that the limited research within the area and the relatively exploratory nature of this study [52].

Mediational analyses were performed to assess the effect of psychological flexibility and depression on the relationship between PTSD, as measured by diagnostic status, the severity of the individual PTSD symptom clusters, and overall PTSD symptom severity from the PDS, and chronic pain, as measured by pain severity and pain interference from the MPI. Four symptom clusters from the emotional numbing model, roughly corresponding to the four

symptom clusters in the DSM-5 PTSD criteria, were used [53-55], i.e. re-experiencing, avoidance, emotional numbing (similar to the negative cognitions and mood factor from the DSM-5), and hyperarousal (similar to the alterations in arousal and reactivity factor from the DSM-5). Psychological flexibility was indexed by total scores on the measures of pain-related acceptance (CPAQ), committed action (CAQ), cognitive fusion (PIPS), and values-based action (CPVI) and depression was measured with the HADS.

Mediation models should be chosen through assumptions that are either invoked based on theory or fulfilled by design features [56]. In accordance with this, our mediation models were based on earlier longitudinal research, which has highlighted the long term impact of PTSD symptoms on pain intensity [14] and has identified mechanisms that mediate the effect of PTSD symptoms on pain intensity [15], and cross-sectional research, which has investigated mediators of the relationship between PTSD and chronic pain [18-21]. The mediation models were also strengthened by the current data, which showed that the trauma producing the current PTSD symptoms happened on average 3-5 years prior to the time that the measures of pain and psychological flexibility were administered. Following recommendations in the literature for mediational studies [17], we also employed an adequately large sample size, measures with sound psychometric properties, and evaluated several mediators simultaneously. As a first step in the mediation analyses, we conducted pairwise correlations between PTSD (diagnostic status, cluster, and overall symptom severity), the measures of psychological flexibility, depression, and chronic pain. Pearson correlation coefficients were calculated, with a point-biserial correlation being used for the dichotomous variable PTSD diagnostic status.

The significance of the indirect effect was estimated using the product of coefficients [57]. This method directly assesses the significance of the indirect or mediating effect. Mediational analyses were tested using the bootstrapping method with bias-corrected confidence estimates

[58]. This method was developed to handle non-parametric data and relatively small sample sizes. The algorithm and syntax for SPSS is available online [59]. To test for individual mediating effects the mediators were first tested separately and then all significant individual mediators were tested simultaneously. Each analysis was based on 5000 bootstrapped samples. The effect size index P_M, which is the ratio of the indirect effect to the total effect, was used to evaluate the magnitude of the indirect effects in the multivariate analysis [60]. All analyses were carried out using SPSS (Version 22) and 2-tailed tests of significance were used for the statistical analyses.

Results

Attrition analyses

Recommended statistical procedures were followed when dealing with missing data [61, 62]. The total percentage of missing values was low (5.1%), whilst 41.3 % of the cases had missing values on at least one item. Little's MCAR test was non-significant (Chi-square = 394.0, df = 396, p = .519), indicating that the data were missing at random and it was deemed appropriate to impute any missing values using the Expectation-Maximization method (EM) [63]. All items were approximately normally distributed based on visual inspection of histograms, normal Q-Q, and box plots. Outliers (n = 3) were identified with the outlier labelling rule using 2.2 as a multiplier and the affected values were winsorized and included in all subsequent analyses [64, 65].

Group comparisons

Table 1 presents descriptive information and statistical comparisons for the demographic variables for the full sample and the subsamples of participants with and without comorbid PTSD. Fibromyalgia (29.2%), cervicocranial syndrome (11.6%), cervicobrachial syndrome (9.6%), lumbago (8.8%), and myalgia (5.6%) were the most frequently reported primary pain diagnoses. The sociodemographic and clinical characteristics of this sample were comparable

to the clinic's referrals as a whole and to people seeking treatment for chronic pain at other regional specialist pain units in Sweden [66].

Descriptive information and statistical comparisons for the dependent variables and the proposed mediators for the full sample and the subsamples of participants with and without comorbid PTSD are displayed in Table 2. In line with expectations, those with PTSD had significantly higher levels of pain severity, pain interference, depression, and cognitive fusion and lower levels of pain-related acceptance and committed action (all p's < .05). No significant difference was found between the groups on values-based action.

Mediation analyses

Mediational analyses were performed to measure the mediating effect of psychological flexibility and depression on chronic pain, using diagnostic status (PTSD and no-PTSD), PTSD symptom severity, and the PTSD symptom clusters as the independent variables. Table 3 presents pairwise correlations between the independent variables (PTSD), the proposed mediators (pain acceptance, committed action, cognitive fusion, values-based action, and depression), and the dependent variables (pain severity and interference) for the full sample. Overall, significant correlations were observed between all variables except for the correlations between the PTSD variables and values-based action as well as the avoidance PTSD cluster and pain severity.

Table 4 presents the results of the univariate mediation analyses using PTSD diagnostic status as the independent variable. The *a*-path represents the effect of PTSD (yes/no) on the mediator and the *b*-path the effect of the mediator on chronic pain controlling for PTSD. The *c*-path represents the total effect of PTSD on chronic pain and the *c´*-path represents the direct effect of PTSD on chronic pain, when controlling for the mediator. The indirect or mediating effect is the effect of the mediator on the relationship between PTSD and chronic pain. The cross-product *a*b* directly assesses the significance of this effect. Confidence intervals are

derived from the obtained distribution of *a*b* scores and the indirect effect is significant at the level specified in the analysis if lower and upper bounds do not contain zero. The cross-product *a*b* corresponds to the difference between the total effect of PTSD on chronic pain and the direct effect of PTSD on chronic pain when controlling for the mediator (*c*-*c´*). The relationship between PTSD and pain severity was mediated (separately) by pain-related acceptance, committed action, cognitive fusion, and depression. The same mediators were significant for the relationship between PTSD and pain interference.

In the next step mediators found to be significant on the univariate level were examined in a multivariate fashion (see Table 5). The relationship between PTSD and pain severity as well as the relationship between PTSD and pain interference were mediated by pain-related acceptance, cognitive fusion, and depression. No mediating effect was seen for committed action. Overall, pain-related acceptance was identified as the strongest mediator from the psychological flexibility model. Mediation analyses using PTSD symptom severity and the PTSD symptom clusters as the independent variables were conducted and comparable levels of variance were explained with consistent result patterns. However, using diagnostic status as the independent variable is in line with similar research [18] and such an approach is preferable over the other available measures since it incorporates not only a wide range of symptoms from all PTSD clusters but also the impact on overall functioning, and only these analyses were retained in the manuscript.

Discussion

The aim of the present study was to examine the relationship between PTSD and chronic pain and in particular whether various indices of psychological flexibility mediate the relationship between these two conditions. First, the findings from this study add to a large body of evidence showing that in people with some form of chronic pain, the presence of PTSD

symptoms is associated with greater adverse impacts on their functioning [6, 15, 18-21, 34]. Participants who reported symptoms consistent with a current diagnosis of PTSD had significantly higher levels of pain severity, pain interference, and depression than those reporting symptoms below diagnostic threshold for PTSD in this study. Second, consistent with a small body of literature looking at psychological flexibility, PTSD, and chronic pain [6, 32-34], participants with PTSD reported lower levels of pain-related acceptance and committed action as well as higher levels of cognitive fusion compared to those without PTSD. No significant difference between these two groups was seen on values-based action.

In terms of mediation it was found that pain-related acceptance, committed action, and cognitive fusion were significant and separate mediators of the relationship between PTSD diagnostic status and chronic pain, as indexed by pain severity and pain interference. Depression was also a significant mediator of the relationship, while no mediating effect was found for values-based action. Overall, the present results suggest that lower levels of acceptance and committed action as well as higher levels of depression and cognitive fusion contribute to more pain severity and pain interference in people with PTSD compared to people without PTSD. When mediators found to be significant on the univariate level were investigated simultaneously cognitive fusion, pain related acceptance, and depression were identified as significant mediators of the relationship between PTSD and chronic pain, with pain-related acceptance being the strongest mediator from the psychological flexibility model.

The results of this study points to experiential avoidance (or low acceptance) as an important mediator of the relationship between PTSD and chronic pain. These results are consistent with those of recent studies that have evaluated pain-related acceptance as a protective psychological variable in trauma-exposed people with chronic pain [6, 33, 34] and shown that acceptance (the opposite of experiential avoidance) is related to fewer psychological symptoms and better outcomes after traumatic exposure [67].

Within a psychological flexibility framework, cognitive fusion is the dysfunctional influence of cognitive content on functioning, or the reduced capacity to respond to contextual stimuli that would better guide behavioral responses in the presence of rigidly held beliefs about pain that people with chronic pain frequently report [68]. The present results suggest that cognitive fusion is an important mediator of the relationship between PTSD and chronic pain. The results are in line with previous studies suggesting that cognitive fusion and similar processes, such as decentering and cognitive inflexibility, are related to functioning in chronic pain and separately to the severity of PTSD [27, 28, 30, 68], and with models of PTSD which specify that the way a person processes and interprets an traumatic event plays an important role in the development and maintenance of symptoms and in response to treatment for PTSD [20, 69, 70]. Furthermore, the results are in accordance with a recent study where catastrophizing and fear-avoidance beliefs, other types of dysfunctional cognitive processes, have been identified as mediating factors of the relationship between PTSD and pain intensity [15]. This is because in contextual terms it is fusion that activates the impact of cognitive content. Overall, the results from this study are consistent with the mutual maintenance model and the fear avoidance model, which suggest that both chronic pain and PTSD are characterized by avoidance and dysfunctional cognitive processes limiting use of adaptive coping [12, 71].

Various models have been applied to explain the relationship between PTSD and chronic pain [18-21], but up until now no studies that we are aware of have investigated whether processes from the psychological flexibility model mediate this relationship. The results from this study suggest that several processes from this model may contribute to the negative interaction between PTSD and chronic pain. Hence, the present study provides important information about these relationships, which strengthen the idea that the psychological flexibility model may help us integrate current knowledge and better understand how PTSD and chronic pain interact to increase the severity of both conditions. These results are consistent with results

from a growing list of studies connecting processes from the theoretically-based and potentially integrative psychological flexibility model to the relationship between PTSD and chronic pain [6, 33, 34] and with studies connecting these processes, or other processes similar to those described in the this model, to the severity of pain and pain-related impairment, and separately to the severity of PTSD [24-31].

Current psychological treatments for chronic pain produce only small to medium effect sizes. Identification of mediators could be used to refine treatment interventions and improve outcomes [72, 73]. The novel findings from this study could be used to guide treatment interventions for PTSD and chronic pain. Possibly, people with these combined problems would benefit from treatment interventions targeting acceptance and other processes from the psychological flexibility model. This suggestion is consistent with results from earlier studies where pain-related acceptance [24, 74-76] and other processes from the psychological flexibility model [25-27, 36, 76] appear to play a role in relation to multiple treatment outcomes in the chronic pain field and particularly in pain populations with relatively high clinical complexity.

There are several limitations to be noted in this study. First, cross-sectional studies can reveal *possible* causal mechanisms especially in studies employing well-founded theories describing the direction of causation and where the cross-sectional data may hold information about temporal process [16, 17]. Theory, recent research and our data, where the trauma producing the current PTSD symptoms was shown to precede the administration of the pain and psychological flexibility measures by on average 3-5 years, guided the arguments made in this study about mediation. Nevertheless, because of the cross-sectional nature of the data, the criterion of temporality where change in the mediator precedes changes in the dependent variable was not met and thus no firm conclusions about the direction of causation can be drawn from this study. The observed relationships from this study provide a basis for further

work aimed at determining the nature of the relation, which may consist of mutual influence, impact of PTSD on chronic pain or vice versa, and longitudinal data are needed to address this issue in future studies. Second, PTSD diagnostic status was based on a self-report measure and not on a semi-structured clinical interview. Third, although there was very little missing data and these data were missing at random our analyses included participants with missing data on one or more of the studied variables. Fourth, the sample characteristics may limit the generalizability of our findings; most participants were women with relatively high levels of education. Fifth, the measure of cognitive fusion used in this study has demonstrated satisfactory psychometric properties [48] and the internal consistency in this sample was satisfactory ($\alpha = .70$) based on established guidelines [77], but potential limitations, such as low internal consistency and incremental validity, have been identified in cross-validation studies after this study began [78, 79].

In conclusion, people seeking treatment for chronic pain who report symptoms consistent with a current diagnosis of PTSD have greater adverse impacts on their functioning than those reporting symptoms below diagnostic threshold for PTSD. Also, processes from the psychological flexibility model were identified as mediators of the relationship between PTSD and these impacts of chronic pain in people seeking treatment for chronic pain. The psychological flexibility model may be useful as an overarching model to help understand the relationship between PTSD and chronic pain. It is possible that targeting pain-related acceptance, committed action, and cognitive fusion (among other processes) in the treatment of chronic pain may produce corresponding improvements in comorbid symptoms of PTSD when these are present and may reduce impacts of PTSD on outcomes of chronic pain. Conversely, targeting of these processes in the treatment of PTSD may produce similar improvements for symptoms of chronic pain. Further research to evaluate these possibilities is needed.

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Table 1 Demographic and clinical characteristics of full sample and participants with and without PTSD

Variable	Full sample (N = 315)	PTSD (N = 127)	No PTSD (N = 188)	*p*
Mean age in years (SD)	41.0 (11.1)	41.7 (11.1)	40.5 (11.0)	.33
% Female	71.1	68.5	72.9	.40
% >12 years education	29.2	27.0	30.6	.49
% Currently working/in education	51.7	51.2	52.0	.90
% Born in Sweden/Nordic country	78.4	74.8	80.9	.20
Mean pain duration in years (SD)	7.5 (7.5)	7.1 (7.4)	7.7 (7.5)	.54
Mean # of pain locations (SD)	15.5 (9.1)	15.9 (9.1)	15.2 (9.0)	.51
Mean PTSD symptom severity (SD)	16.9 (12.4)	27.3 (10.2)	9.8 (8.2)	.00
Time since traumatic event:				.08
% 0-6 months	5.3	6.5	4.5	
% 6 months to 3 years	21.5	24.2	19.6	
% 3 to 5 years	12.5	16.9	9.5	
% > 5 years	60.7	52.4	66.5	
Primary traumatic event:				.10
% Accident	23.8	27.6	21.3	
% Non-sexual assault	8.9	6.3	10.6	
% Sexual assault	13.0	9.4	15.4	
% Other traumatic event	31.4	29.9	32.4	
% Life threatening illness	8.3	7.1	9.0	
% Multiple traumas	14.6	19.7	11.2	

Note: PTSD = Posttraumatic Stress Disorder; PTSD symptom severity measured using the Posttraumatic Diagnostic Scale (1-10 = mild; 11-20 = moderate; 21-35 = moderate to severe; > 36 = severe).

Table 2 Means, standard deviations and statistical comparisons for mediator and dependent variables for participants with and without PTSD

Clinical characteristics	Full sample (N=315)	PTSD (N=127)	No PTSD (N=188)	t-value	Cohen's d
Mediators					
Pain acceptance	41.83 (17.50)	37.17 (17.43)	44.98 (16.87)	-3.98**	.46
Committed action	59.45 (16.41)	53.76 (17.12)	63.28 (14.77)	-5.11**	.60
Cognitive fusion	22.95 (4.16)	23.64 (3.62)	22.48 (4.44)	2.55*	-.28
Values-based action	2.05 (.96)	1.94 (1.05)	2.13 (.88)	-1.72	.20
Depression	10.22 (4.60)	11.81 (4.40)	9.15 (4.43)	5.25**	-.60
Dependent variables					
Pain severity	4.73 (.85)	4.89 (.78)	4.62 (.88)	2.75**	-.32
Pain interference	4.76 (.92)	5.00 (.88)	4.59 (.91)	3.97**	-.46

Notes: PTSD = Posttraumatic Stress Disorder; Mediators were measured using the Chronic Pain Acceptance Questionnaire, Committed Action Questionnaire, Psychological Inflexibility in Pain Scale, and Chronic Pain Values Inventory respectively; Dependent variables were measured using the Multidimensional Pain Inventory. Levene's test for equality of variances indicated unequal variances between the groups on committed action and cognitive fusion and the statistics were adjusted accordingly. Degrees of freedom=243-313.
*p < .05, * *p < .01

Table 3 Pairwise correlations between the independent (PTSD), mediator (psychological flexibility) and dependent variables (chronic pain) for the full sample (N = 315)

	1	2	3	4	5	6	7	8	9	10	11	12	13
Independent variables													
1. PTSD diagnosis	1												
2. PTSD symptom severity	.69**	1											
3. PTSD re-experiencing	.58**	.87**	1										
4. PTSD avoidance	.49**	.71**	.62**	1									
5. PTSD numbing	.61**	.87**	.65**	.67**	1								
6. PTSD hyperarousal	.57**	.85**	.60**	.45**	.63**	1							
Mediators													
7. Pain acceptance	-.22**	-.29**	-.21**	-.18**	-.31**	-.23**	1						
8. Committed action	-.29**	-.41**	-.33**	-.20**	-.36**	-.38**	.35**	1					
9. Cognitive fusion	.14*	.24**	.19**	.16**	.23**	.21**	-.54**	-.17**	1				
10. Values-based action	-.10	-.02	.03	.06	-.06	-.02	.27**	.23**	-.12*	1			
11. Depression	.29**	.39**	.33**	.12*	.38**	.36**	-.51**	-.44**	.28**	-.37**	1		
Dependent variables													
12. Pain severity	.15**	.23**	.24**	.10	.17**	.18**	-.46**	-.17**	.41**	-.12*	.41**	1	
13. Pain interference	.22**	.28**	.22**	.13*	.31**	.23**	-.61**	-.26**	.43**	-.27**	.58**	.60**	1

Notes: Independent variables were measured using the Posttraumatic Diagnostic Scale; Mediators were measured using the Chronic Pain Acceptance Questionnaire, Committed Action Questionnaire, Psychological Inflexibility in Pain Scale, and Chronic Pain Values Inventory respectively; Dependent variables were measured using the Multidimensional Pain Inventory.
*p < .05; * *p < .01

Table 4 Results of the univariate mediator analyses between PTSD diagnostic status and pain severity and pain interference

Dependent variable	Mediator	Total Direct & Indirect effects		Results for Indirect Effects *a*b*	
		Path	Point-Estimate (SE)	Point-Estimate (SE)	95% CI LL, UL
Pain severity					
	Pain acceptance	a	-7.81* (1.96)	.17* (.05)	.08, .28
		b	-.02* (.00)		
	Committed action	a	-9.52* (1.81)	.07* (.03)	.01, .14
		b	-.01* (.00)		
	Cognitive fusion	a	1.16* (.47)	.09* (.04)	.02, .19
		b	.08* (.01)		
	Values-based action	a	-.19 (.11)	.02 (.02)	-.00, .07
		b	-.09 (.05)		
	Depression	a	2.67* (.51)	.19* (.05)	.11, .30
		b	.07* (.01)		
Pain interference					
	Pain acceptance	a	-7.81* (1.96)	.24* (.06)	.12, .37
		b	-.03* (.00)		
	Committed action	a	-9.52* (1.81)	.12* (.04)	.06, .21
		b	-.01* (.00)		
	Cognitive fusion	a	1.16* (.47)	.10* (.04)	.02, .19
		b	.09* (.01)		
	Values-based action	a	-.19 (.11)	.04 (.03)	-.00, .11
		b	-.24* (.05)		
	Depression	a	2.67* (.51)	.30* (.06)	.19, .43
		b	.11* (.01)		

Notes: LL = lower limit; UL = upper limit. Unstandardized coefficients are reported and the indirect effect is statistically significant if the confidence interval (CI) does not include zero. Asterisks (*) indicate a statistically significant effect.

Table 5 Results of the multivariate mediator analyses between PTSD diagnostic status and pain severity and pain interference

Dependent Variable	Mediator	Total Direct & Indirect Effects		Results for Indirect Effects *a*b*		
		Path	Point-Estimate (SE)	Point-Estimate (SE)	95% CI LL, UL	P_M, (ab)/c
Pain severity						
		Total c	.27* (.10)			
		Direct c´	.03 (.09)			
	Pain acceptance	a	-7.81* (1.96)	.09* (.04)	.03, .18	.33
		b	-.01* (.00)			
	Committed action	a	-9.52* (1.81)	-.03 (.03)	-.10, .02	ns
		b	.00 (.00)			
	Cognitive fusion	a	1.16* (.47)	.05* (.03)	.01, .12	.19
		b	.05* (.01)			
	Depression	a	2.67* (.51)	.12* (.04)	.06, .21	.44
		b	.05* (.01)			
Pain interference						
		Total c	.41* (.10)			
		Direct c´	.06 (.08)			
	Pain acceptance	a	-7.81* (1.96)	.15* (.05)	.07, .25	.37
		b	-.02* (.00)			
	Committed action	a	-9.52* (1.81)	-.03 (.02)	-.09, .01	ns
		b	.00 (.00)			
	Cognitive fusion	a	1.16* (.47)	.03* (.02)	.00, .09	.07
		b	.03* (.01)			
	Depression	a	2.67* (.51)	.20* (.05)	.12, .30	.49
		b	.07* (.01)			

Notes: LL = lower limit; UL = upper limit. Unstandardized coefficients are reported and the indirect effect is statistically significant if the confidence interval (CI) does not include zero. Asterisks (*) indicate a statistically significant effect.