Dimensions of catastrophic thinking associated with pain experience and disability in patients with neuropathic pain conditions

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Abstract

The objective of the present study was to examine the relative contributions of different dimensions of catastrophic thinking (i.e. rumination, magnification, helplessness) to the pain experience and disability associated with neuropathic pain. Eighty patients with diabetic neuropathy, post-herpetic neuralgia, post-surgical or post-traumatic neuropathic pain who had volunteered for participation in a clinical trial formed the basis of the present analyses. Spontaneous pain was assessed with the sensory and affective subscales of the McGill Pain Questionnaire. Pinprick hyperalgesia and dynamic tactile allodynia were used as measures of evoked pain. Consistent with previous research, individuals who scored higher on a measure of catastrophic thinking (Pain Catastrophizing Scale; PCS) also rated their pain as more intense, and rated themselves to be more disabled due to their pain. Follow up analyses revealed that the PCS was significantly correlated with the affective subscale of the MPQ but not with the sensory subscale. The helplessness subscale of the PCS was the only dimension of catastrophizing to contribute significant unique variance to the prediction of pain. The PCS was not significantly correlated with measures of evoked pain. Catastrophizing predicted pain-related disability over and above the variance accounted for by pain severity. The findings are discussed in terms of mechanisms linking catastrophic thinking to pain experience. Treatment implications are addressed.

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Keywords: Catastrophizing; Helplessness; Neuropathic pain; Affective pain

1. Introduction

There has been increased interest in understanding psychological influences on the experience of neuropathic pain (Haythornthwaite and Benrud-Larson, 2000). Research suggests a relation between pain catastrophizing and neuropathic pain, but it remains unclear which dimensions of pain catastrophizing contribute to heightened neuropathic pain. It is also unclear whether pain catastrophizing is associated with both spontaneous and evoked pain in patients with neuropathic pain conditions. More in-depth investigations of the relation between pain catastrophizing and neuropathic pain might elucidate the mechanisms linking catastrophic thinking to neuropathic pain experience and might highlight potential targets of intervention.

1.1. The psychology of neuropathic pain

Neuropathic pain refers to pain ‘initiated or caused by a primary lesion or dysfunction in the nervous system’ (Merskey and Bogduk, 1994). Neuropathic pain can arise as a result of dysfunction of either peripheral or central neural mechanisms (Attal and Bouhassira, 1999; Devor and Seltzer, 1999). Spontaneous and evoked pain are distinct forms of pain experience associated with neuropathic pain conditions (Bridges et al., 2001; Dworkin, 2002). Spontaneous pain is experienced on a persistent or intermittent basis with or without stimulation of the affected area; evoked pain is experienced only upon stimulation of the affected area. Research has yet to examine the relation between psychological variables and evoked pain. Since spontaneous and evoked pain may be mediated by distinct mechanisms, differential relations between psychological variables and spontaneous and evoked pain might clarify the processes...
linking psychological and physiological mechanisms of pain experience (Dworkin, 2002; Gracely et al., 2004).

Pain catastrophizing has been shown to be a determinant of pain-related outcomes associated with neuropathic pain (Haythornthwaite and Benrud-Larson, 2000; Turner et al., 2002). Pain catastrophizing has been defined as a negative cognitive set brought to bear during actual or anticipated pain experience (Sullivan et al., 2001). Research has supported a multidimensional conceptualization of pain catastrophizing, comprising elements of rumination, magnification and helplessness (Osman et al., 2000; Sullivan et al., 1995, 2000). Pain catastrophizing has been associated with heightened pain experience in patients with post-herpetic neuralgia (Haythornthwaite et al., 2003), phantom limb pain (Jensen et al., 2002), and pain associated with spinal cord injury (Turner et al., 2002). Research however has yet to identify the specific dimensions of pain catastrophizing that are associated with neuropathic pain. Specification of the dimensions of pain catastrophizing associated with neuropathic pain might have implications for the development of targeted interventions aimed at facilitating adjustment to, or promoting recovery from, neuropathic pain conditions.

1.2. The present research

The present research examined the relation between specific components of pain catastrophizing (i.e. rumination, magnification, helplessness) and the pain experience associated with neuropathic pain conditions. Also of interest was whether components of pain catastrophizing were differentially related to measures of spontaneous and evoked pain. Individuals diagnosed with one of three neuropathic pain conditions completed measures of pain catastrophizing, spontaneous pain, evoked pain and pain-related disability. Regression analyses were used to assess the unique and shared contributions of different components of pain catastrophizing to the prediction of pain-related outcomes.

2. Methods

2.1. Participants

Eighty patients with diabetic neuropathy (n = 19), post-herpetic neuralgia (n = 12) or post-surgical or post-traumatic neuropathic pain (n = 49) who had volunteered for participation in a clinical trial formed the basis of the present analyses. The mean age of participants was 52.5 years with a range of 24–84 years. Mean duration of pain was 69 months (SD = 68.7).

2.2. Procedure

Participants were recruited from three hospital outpatient pain management units in eastern Canada between September 2001 and December 2002. All participants were examined by pain specialists to confirm the diagnosis of neuropathic pain. Participants were asked to complete measures of current spontaneous pain (i.e. pain symptoms associated with their pain condition), pain catastrophizing and functional disability during the intake assessment. One of three physicians performed evaluations of dynamic tactile allodynia and pinprick hyperalgesia. All measures were completed on the same day. These evaluations were conducted as part of a baseline assessment for a clinical trial of three topical analgesic creams (ketamine, amitriptyline, ketamine + amitriptyline) for the treatment of neuropathic pain. Participants were permitted to continue their regular medication regimen. Results pertaining to the outcome of the clinical trial are described elsewhere (Lynch et al., 2004).

2.2.1. Measures

2.2.1.1. Spontaneous pain. The short form McGill Pain Questionnaire (MPQ; Melzack, 1987) was used as a measure of current spontaneous pain. On this measure, respondents are asked to rate the quality and intensity (e.g. cramping, hot, achng, cruel) of their pain on 15 different 4-point Likert-scales with the endpoints (0) not at all and (3) severe. Values were summed to yield subscale scores for the sensory and affective dimensions of pain experience. The MPQ has been shown to be one of the most reliable measures of chronic pain experience (Melzack, 1987; Turk et al., 1985).

2.2.2. Evoked pain

Measures of evoked pain included dynamic tactile allodynia and pinprick hyperalgesia. During stimulation, participants rated their pain on a 100 mm visual analog scale (VAS). Dynamic tactile alodynia was assessed by light-touch dynamic stimulation of the site of pain. A one-inch wide foam brush was applied to the site of pain in 3–5 cm strokes of 1 s duration (Dworkin, 2002). Pinprick hyperalgesia was assessed by applying an autoclaved standardized safety pin to the painful site and to a corresponding normal site (typically contralateral). Participants were asked to compare the sensations of the pinprick on the affected and normal sites, and to rate the severity of pain at the affected site (Dworkin, 2002).

2.2.3. Pain catastrophizing

The Pain Catastrophizing Scale (Sullivan et al., 1995) was used to measure catastrophic thinking related to pain. The PCS consists of 13 items describing different thoughts and feelings that individuals might experience when they are in pain. Responses were summed to yield three different subscales: rumination, magnification and helplessness. The PCS has been shown to have high internal consistency (coefficient alpha = 0.87), and to be associated with heightened pain, disability as well as employment status (Sullivan et al., 1995; Sullivan et al., 1998; Sullivan and Stanish, 2003).

2.2.4. Functional disability

The Pain Disability Index (Pollard, 1984) assesses the degree to which respondents perceive themselves to be disabled in seven different areas of daily living (home, social, recreational, occupational, sexual, self-care, life support). For each life domain, respondents are asked to provide perceived disability ratings on 11-point scales with the endpoints (0) no disability and (10) total disability. The PDI has been shown to be internally reliable
and significantly correlated with objective indices of disability (Tait et al., 1987, 1990).

3. Results

Characteristics of the sample are presented in Table 1. Significant differences across diagnostic subgroups were obtained for age, $F(2,77)=23.0$, $P<0.001$, and dynamic tactile allodynia, $F(2,77)=3.7$, $P<0.01$. As shown in Table 1, participants with post-herpetic neuralgia were significantly older than participants with diabetic neuropathy, who in turn were significantly older than participants with post-surgical or post-traumatic neuropathy. Participants with diabetic neuropathy provided lower ratings of dynamic tactile allodynia than participants in the other two diagnostic subgroups. A significant sex difference was found for hyperalgesia, $t(78)=3.0$, $P<0.01$, where women rated their sensations to pinprick stimulation as more intense than men. A marginally significant sex difference was obtained for dynamic tactile allodynia, $t(78)=1.8$, $P=0.06$, where women again reported greater pain intensity than men.

Group means on measures of pain intensity and catastrophic thinking are similar to those reported in samples of individuals with chronic musculoskeletal pain (Sullivan et al., 1998; Sullivan and Stanish, 2003). Group means for functional disability associated with pain are slightly lower than those obtained in samples of individuals with chronic musculoskeletal pain (Tait et al., 1990; Sullivan et al., 1998).

3.1. Catastrophizing and spontaneous pain

Consistent with previous research, the total PCS was significantly correlated with the severity of pain symptoms (MPQ PRI) associated with neuropathic pain, $r=0.25$, $P<0.05$. Analyses further revealed that the total PCS was significantly correlated with the affective subscale of the MPQ, $r=0.42$, $P<0.001$, but not with the sensory subscale of the MPQ, $r=0.11$, ns.

A hierarchical regression analysis was conducted to assess the relative contribution of the three subscales of the PCS to the prediction of the affective subscale of the MPQ. Age and sex were entered in the first step of the analysis and contributed less than 1% of variance to the prediction of affective pain, $F(2,77)=.27$, ns. The rumination, magnification and helplessness subscales of the PCS were entered in the second step, contributing an additional 29% of variance to the prediction of affective pain, $F$ change (3,74) = 10.4, $P<0.001$. Examination of the beta weights for the final regression equation revealed that only the helplessness subscale of the PCS contributed significant unique variance to the prediction of affective pain (Table 2).

A second hierarchical regression was conducted in order to determine whether the observed relations between PCS subscales and affective pain were constant across diagnostic groups. Interaction terms were computed by multiplying dummy coded variables for each diagnostic group with the different PCS subscales. Interaction terms were included as the last step in the regression analysis. The inclusion of the interaction terms did not contribute significant variance to the prediction of affective pain, $F$ change (3,71) = 0.92, ns. In other words, the relation between PCS subscales and affective pain was not moderated by diagnostic group.

3.2. Catastrophizing and evoked pain

Correlational analyses revealed that the total PCS was not significantly correlated with measures of dynamic tactile allodynia, $r=0.12$, ns, or pinprick hyperalgesia, $r=0.09$, ns. Follow-up analyses revealed that none of the PCS subscales were significantly correlated with dynamic tactile allodynia or hyperalgesia.

3.3. Catastrophizing and disability associated with neuropathic pain

A hierarchical regression was conducted to assess the relative contribution of the three subscales of the PCS to the prediction of self-reported functional disability due to pain. Age and sex were entered in the first step of the analysis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>$R$</th>
<th>$F$ change</th>
<th>$P$</th>
<th>$r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
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<td>$0.05$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>$-0.05$</td>
<td>$0.06$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rumin</td>
<td>$0.18$</td>
<td>$0.28**$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magni</td>
<td>$0.04$</td>
<td>$0.20*$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Helps</td>
<td>$0.55$</td>
<td>$0.50**$</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: $N=80$. Dependent variable is the affective subscale of the MPQ. Rumin, rumination subscale of the PCS. Magni, magnification subscale of the PCS. Helps, helplessness subscale of the PCS. *$P<0.05$, ** $P<0.01$. Beta weights are from the final regression equation.
and contributed less than 1% of variance to the prediction of affective pain, $F(2,77) = 0.09$, ns. The MPQ PRI was entered in the second step of the analysis and contributed an additional 8% of variance to the prediction of self-rated disability, $F \text{ change} (1,76) = 6.4$, $P < 0.01$. The rumination, magnification and helplessness subscales of the PCS were entered in the third step, contributing an additional 13% of variance to the prediction of functional disability, $F \text{ change} (3,73) = 4.0$, $P < 0.01$. Although the PCS subscales as a group were predictive of functional disability, none of the subscales contributed significant unique variance to the prediction of self-rated disability (Table 3). A follow-up regression analysis revealed the relation between the PCS and functional disability was not moderated by diagnostic group.

### 4. Discussion

The findings of the present study join a growing literature suggesting that catastrophic thinking is significant determinant of pain experience associated with neuropathic pain (Haythornthwaite et al., 2003; Jensen et al., 2002). The present study extends previous research in showing that, of the different components of pain catastrophizing, helplessness is most strongly associated with the experience of neuropathic pain. Specific neuropathic diagnosis did not moderate the relation between the helplessness dimension of catastrophizing and pain intensity, suggesting that the relation between catastrophizing and neuropathic pain may be observed across various types of neuropathic pain conditions.

Research on the differential relation between dimensions of pain catastrophizing and pain-related outcomes has been driven by theoretical and applied considerations. From a theoretical perspective, there has been interest in bringing greater specificity to the mechanisms linking psychological and physiological processes of pain perception (e.g. Sullivan et al., 2001). From an applied perspective, research has been motivated by the pursuit for more effective targeted interventions for individuals suffering from persistent and debilitating pain conditions (Thorn et al., 2002; Sullivan and Stanish, 2003).

In the present study, the helplessness dimension of pain catastrophizing emerged as the strongest predictor of spontaneous pain. It is possible, that the enduring trajectory of neuropathic conditions and relative inefficacy of available treatments may create a life context that might entrain a helpless orientation toward dealing with pain. In turn, a helpless orientation to dealing with pain might compromise efforts to invoke coping strategies for pain (e.g. Crisson and Keefe, 1988). Alternatively, a helpless orientation to pain might be associated with the use of other passive or avoidant strategies that might contribute to heightened pain (Brown and Nicassio, 1987). The persistent expression of helplessness may also begin to erode the interpersonal support resources of the individual with neuropathic pain (Cano et al., 2004; Lackner and Gurtman, 2004). A recursive process may then become established where a helpless orientation to pain contributes directly or indirectly to increased pain, which then further accentuates feelings of helplessness.

Previous research addressing the relation between pain catastrophizing and neuropathic pain has used the catastrophizing subscale of the CSQ (Haythornthwaite et al., 2003; Jensen et al., 2002). It is noteworthy that the helplessness subscale of the PCS contains five of the six items that make up the catastrophizing subscale of the CSQ. In this regard, the relation between helplessness and pain catastrophizing observed in the present study is consistent with previous research.

In previous research with musculoskeletal pain samples, the rumination subscale of the PCS has tended to show the strongest association with pain experience (Sullivan et al., 1998). Explanations of the relation between rumination and pain experience have appealed to attentional mechanisms that might contribute to heightened pain experience (Crombez et al., 1998; Eccleston and Crombez, 1999; Peters et al., 2000). In the present research, the zero order correlation between rumination and pain experience was significant but was no longer significant when helplessness was statistically controlled. Thus, it is unlikely that attentional mechanisms were the primary determinants of increased pain experienced by individuals who obtained high catastrophizing scores. In a previous study addressing the relation between catastrophizing and chronicity, Sullivan et al. (2002a,b,c) reported that the helplessness scale of the PCS was the strongest predictor of pain experience only in the subgroup of the most chronic pain patients (pain duration greater than 4 years). Viennet et al. (1999) also reported that the helplessness component of catastrophizing was most strongly associated with pain experience in a very chronic sample of pain patients (mean duration of pain of 7.5 years). These findings suggest that the relation between helplessness and pain experience may not be specific to neuropathic pain, but may emerge as

### Table 3

<table>
<thead>
<tr>
<th>Variable</th>
<th>$R$</th>
<th>Beta</th>
<th>$F$ change</th>
<th>$P$</th>
<th>$r$</th>
</tr>
</thead>
<tbody>
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<td>Sex</td>
<td>0.09</td>
<td>0.04</td>
<td>0.09 (2.77)</td>
<td>0.91</td>
<td>0.01</td>
</tr>
<tr>
<td>Age</td>
<td>0.04</td>
<td>0.03</td>
<td>0.09 (2.77)</td>
<td>0.91</td>
<td>0.01</td>
</tr>
<tr>
<td>MPQ PRI</td>
<td>0.29</td>
<td>0.16</td>
<td>6.4 (1.76)</td>
<td>0.01</td>
<td>0.28**</td>
</tr>
<tr>
<td>Rumin</td>
<td>0.07</td>
<td>0.07</td>
<td>0.33**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magni</td>
<td>0.03</td>
<td>0.22*</td>
<td>6.4 (1,76)</td>
<td>0.01</td>
<td>0.42**</td>
</tr>
<tr>
<td>Helps</td>
<td>0.46</td>
<td>0.29</td>
<td>3.8 (3.73)</td>
<td>0.01</td>
<td>0.42**</td>
</tr>
</tbody>
</table>

Note: $N=80$. Dependent variable is the PDI. Rumin, rumination subscale of the PCS, Magni, magnification subscale of the PCS, Helps, helplessness subscale of the PCS. * $P < .05$, **$P < .01$. Beta weights are from the final regression equation.
a consequence of numerous years of suffering from a debilitating pain condition (Sullivan et al., 2001).

Follow up analyses revealed that pain catastrophizing was associated with the affective but not the sensory subscale of the MPQ. This finding suggests that pain catastrophizing might be differentially linked to affective and sensory dimensions of pain. Consistent with these findings, Geisser et al. (1994) reported that the catastrophizing subscale of the CSQ was correlated with the affective and evaluative subscales of the MPQ, but not the sensory subscale. In a recent study examining the central anatomical correlates of pain in fibromyalgia, Gracely et al. (2004) found that high pain catastrophizing was uniquely associated with activation of brain areas associated with emotion, attention and motor control (e.g. contralateral anterior ACC, contralateral and ipsilateral lentiform). Taken together, these findings suggest that the mechanisms linking pain catastrophizing to pain experience might be specific to the affective dimensions of pain. Few studies on pain catastrophizing have used measures of pain that yield sensory and affective scores and as such the robustness of the current finding is uncertain. However, given the important theoretical implications of the specificity of the relation between pain catastrophizing and the affective dimension of pain, it is recommended that future research in this area use multidimensional measures of pain experience.

No significant association was found between pain catastrophizing and evoked pain ratings. One possibility is that the physiological processes involved in allodynia and hyperalgesia are distinct from the mechanisms associated with spontaneous pain and may have different psychological correlates. In a related manner, evoked pain may tap primarily sensory dimensions of pain which may share little variance with pain catastrophizing. Price and Bushnell (2004) have proposed that nociceptive sensations are processed prior to the implication of psychological mechanisms associated with the appraisal of threat and the experience of negative affect; the latter more likely to be influenced by memory or experience-based beliefs. The spontaneous pain symptoms of individuals with neuropathic pain conditions might be intricately connected to a network of memories and affects associated with the negative impact of pain on day-to-day life. Evoked pain stimuli may not lead to activation of these networks to the same extent as spontaneous pain.

Another possibility is that the evoked pain stimuli (e.g. pinprick, foam brush) were not interpreted by participants as threatening stimuli. Not only are these stimuli not typically considered as pain inducing stimuli, they were used in the context of a medical assessment. In previous research using non-noxious stimuli, relations between pain catastrophizing and perception of unpleasant sensations have been of modest magnitude or non-significant (Peters et al., 2000; Sullivan et al., 2004). It is possible that the psychological processes implicated in catastrophizing effects on pain may require a critical level of stress or threat in order to be activated (Sullivan et al., 2001).

The results of the present study extend previous findings in showing that pain catastrophizing might be a determinant of disability associated with neuropathic pain. Although the focus of research to date has been on the psychological correlates of neuropathic pain experience, the disability that accompanies pain may have an even more pronounced impact on quality of life. Regression analyses revealed that pain catastrophizing contributed to the prediction of functional disability beyond the variance accounted for by pain intensity. These findings suggest that treatment interventions aimed at reducing catastrophic thinking might lead to meaningful reductions in the level of disability associated with neuropathic pain. The findings also suggest that psychological interventions that specifically target catastrophic thinking (e.g. Burns et al., 2003; Thorn et al., 2002; Sullivan and Stanish, 2003) might be usefully incorporated in treatment programs for individuals suffering from neuropathic pain conditions.

A number of design limitations caution the nature of conclusions that can be drawn from this study. First, the cross-sectional design precludes statements about the causal relations between pain catastrophizing, pain experience and pain-related disability. More confident statements about the causal or antecedent status of pain catastrophizing in relation to neuropathic pain outcomes would require replication within a prospective design (Haythornthwaite et al., 2003). More confident statements about differential mechanisms implicated in spontaneous and evoked pain will also require examination of sensory and affective dimensions of both types of stimuli. The data used for the analyses presented in this paper were from a sample of individuals who had volunteered for participation in a clinical trial and therefore may not be representative of the population of individuals with neuropathic pain. In spite of these limitations, the results suggest that pain catastrophizing is a psychological variable that might be specifically associated with the affective dimension of neuropathic pain experience, and that the inclusion of interventions that aim to reduce pain catastrophizing might yield more positive outcomes for individuals being treated for neuropathic pain.

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References

Peters ML, Vlaeyen JWS, Van Druenen C. Do fibromyalgia patients display hypervigilance for innocuous sensory stimuli? Application of a body scanning reaction time paradigm Pain 2000;86:283–92.