The role of catastrophising in rheumatic diseases

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ABSTRACT

Pain is a common and debilitating symptom of many rheumatic diseases. Decades of research have shown that psychological factors are critical in shaping the experience of acute and chronic pain. The current review focuses on pain catastrophising, a cognitive and emotional response to pain, and its implication for the assessment and treatment of individuals with rheumatic diseases. Catastrophising is one of the most salient psychosocial predictors of a host of pain-related outcomes including heightened disability and depression, increased use of opioids, reduced response to treatment and increased chance of long-term postoperative pain. Despite being stable without treatment, catastrophising can be reduced though psychological and non-psychological interventions. Mechanisms of action including physiological, cognitive-behavioural, and social routes are discussed and a comprehensive developmental model of the combined effects of catastrophising, its biological effects and pain is suggested. Identifying patients at risk for poor outcomes through the assessment of catastrophising could enable providers to individually tailor treatment plans and improve clinical outcomes.

***"Pain is inevitable, suffering is optional"* (Dalai Lama XIV)**

Rheumatic diseases are most commonly characterised by chronic pain, yet there is often a discrepancy between more objective measures of disease severity, such as radiography or physician ratings and the patient's report of their pain and disability (1, 2). A host of psychosocial factors contribute to the shaping of the pain experience and the manner in which the individual adjusts to it (3). Catastrophising is an example of a pain-specific psychological construct with a robust impact on pain and related outcomes. Catastrophising was originally conceptualised as a general tendency to magnify and exaggerate the response to threat (4). In the context of pain, it is a set of negative cognitive and emotional responses triggered by actual or expected pain (5, 6) that derives from both genetic and learned/environmental factors (7). A number of comprehensive reviews have summarised the strong evidence for the role of catastrophising in rheumatic diseases such as rheumatoid arthritis, osteoarthritis arthritis, fibromyalgia and scleroderma (8, 9). Since the publications of these reviews, numerous studies continue to demonstrate the deleterious effect of catastrophising on the experience and adjustment to pain in a variety of medical conditions. The current review will briefly summarise key points presented in previous reviews and focus on new and important finding published since 2010 on the relationship between catastrophising and rheumatic diseases. For example, catastrophising has been recently associated with long term pain and disability in osteoarthritis (10), predicts increased pain following total knee arthroplasty (11, 12) and postoperative pain following a variety of surgeries (13). High catastrophising might be especially detrimental to individuals with rheumatoid arthritis who have a low level of education and low social functioning (14), and catastrophising has a greater impact on pain interference and physical health status when pain intensity is low (15).

Pain catastrophising is most commonly assessed through two self-report questionnaires: The Pain Catastrophising Scale (16) and the catastrophising subscale of the Coping Strategies Questionnaires (CSQ) (17). These validated scales ask participants to rate the degree they experienced thoughts and feelings regarding past pain experiences. This captures a general trait-like or dispositional measure of catastrophising, which has been studied for more than two decades. More recently, pain catastrophising has also been assessed as a reaction to a specific situation, usually

experimental pain or pain experienced during a specific time frame as in daily diaries, resulting in a situational measure of catastrophising. Interestingly, these two approaches to measuring pain catastrophising may not be correlated, particularly in individuals who do not experience ongoing pain (*i.e.*, "healthy volunteers") and they vary in their ability to predict pain in different populations and circumstances (18, 19). For example, situational but not dispositional catastrophising predicted postoperative pain in pain-free patients (20).

Longitudinal studies show that dispositional catastrophising is stable over time in multiple groups experiencing persistent pain including rheumatoid arthritis (21), fibromyalgia (22), joint pain (23), and post-herpetic neuralgia (24). Dispositional catastrophising is also stable across weeks in healthy individuals following resolution of acute tooth pain (25). Research suggests that dispositional catastrophising in not a good predictor of laboratory pain, in general; however, it is useful for predicting clinical pain which is critical in rheumatic diseases.

An important issue that arises in any discussion of catastrophising is the question of whether pain elicits worsening catastrophising or does catastrophising contribute to worsening pain? In general, the research currently available support the latter: catastrophising contributes to worsening pain more consistently than pain appears to elicit worsening catastrophising (19). In clinical samples, early changes in catastrophising predict later improvement in clinical pain after an intervention focusing on lifestyle and physical activity for fibromyalgia patients (22). This finding, seen in other patients with chronic pain (26), suggests that reductions in catastrophising are one mechanism involved in pain relief.

Changing catastrophising

Despite the evidence of the stability of catastrophising, it is malleable to different types of treatments. Psychological interventions geared towards improving pain management and reducing catastrophising through teaching patients to identify and replace negative thoughts and cognitions with more adaptive thinking and coping strategies (27) are effective in reducing catastrophising in a variety of pain conditions (28) including rheumatic diseases (29, 30). Even CBT interventions designed to improve sleep in patients with knee osteoarthritis significantly reduced catastrophising (31).

More interestingly, non-psychological interventions can also significantly improve catastrophising, including resistance exercise training (32), oral splints in patient with temporomandibular joint disorder (33), and even medication such as gabapentin (34) and opioids (35) reduce the helplessness component of catastrophising. The sensitivity of catastrophising to a variety of both psychological and non-psychological interventions suggests that it can be improved upon through both direct and indirect mechanisms.

Treatment response

One of the most clinically important aspects of catastrophising is its association with poor outcomes of a variety of treatments for pain, including psychological interventions (36). Patients with low back pain who are high in catastrophising experience worse health related outcomes (37) and reductions in catastrophising mediate their response to many types of physical therapy and behavioural treatments (38). High catastrophising has been associated with increased use of opioids after total knee replacement surgery (39) and surgery for musculoskeletal trauma (40). It is also associated with poor pain relief from various pharmacological treatments including opioids and gabapentin in a variety of chronically painful conditions (41, 42). Additionally, higher catastrophising scores predict worse outcomes following other medical interventions such as radiofrequency neurotomy for chronic whiplash (43), spinal cord stimulation for multiple pain conditions (44), and transcutaneous electrical nerve stimulation after total knee arthroplasty (45).

Mechanisms underlying catastrophising

There are a number of pathways by which catastrophising is thought to influence pain and related outcomes such as disability. These include physiological, cognitive-behavioural, and social routes of action; for a more in-depth discussion, the reader is referred to a comprehensive review by Edwards and colleagues (9).

Physiological

Both central and peripheral mechanisms are thought to be involved the influence of catastrophising on pain. Catastrophising and other psychological factors correlate with endogenous opioid function (46), which may be one of the mechanisms by which catastrophising influences opioid analgesia (42). Modulation of pain by the central nervous system involves multiple pathways and recent interest in central sensitisation (47), a key feature in fibromyalgia (48), has been documented in osteoarthritis (49). Early studies in healthy women showed an association between high catastrophising and greater temporal summation (50), an indicator of central sensitisation. Recent work reports a similar relationship in chronic pain populations (51), and laboratory manipulation of catastrophising increases central sensitisation to pain in women with chronic back pain (52). Alterations in brain resting state and activation by pain in specific brain regions is associated with catastrophising (see Malfliet for a recent review (53)). Interestingly, increased prefrontal cortex gray matter were associated with reductions in catastrophising following cognitive-behavioural therapy in a mixed sample of chronic pain patients (54). More evidence shows that reductions in catastrophising may modulate pain at the superspinal rather than spinal level (55), and a role for peripheral mechanisms is indicated by the association between high catastrophising and elevated cortisol levels in response to laboratory pain testing (56) as well as to interleukin-6 (IL-6) reactivity (57).

Cognitive-behavioural

Some of the earliest conceptual work on pain catastrophising derived from the fear avoidance model (58) in which catastrophising thoughts/beliefs elicited by pain promote fear and (future)

avoidance of pain, thereby contributing to enhanced pain and increased disability. Laboratory studies indicate that even healthy individuals who are high in catastrophising have difficulty disengaging from the threat of pain (59). More recent work in healthy individuals high in catastrophising indicates that this difficulty to disengage delays the efficacy of other coping skills, such as distraction (60); however, distraction may be beneficial to chronic pain populations high in catastrophising (61). Catastrophising can also significantly impact the type of behaviours individuals engage in, particularly health related behaviours. For example, in fibromyalgia, high catastrophising is correlated with a reduced ability to participate in meaningful activities and the perception of greater functional impairment (62). Similarly, in back pain, high catastrophising was associated with longer periods of bed rest (63). Another obstacle for engaging in physical activity is fatigue, a common symptom of rheumatic diseases that is also significantly associated with catastrophising (64). These effects of catastrophising on behaviour can act as perpetuating factors in the vicious cycle of pain and disability.

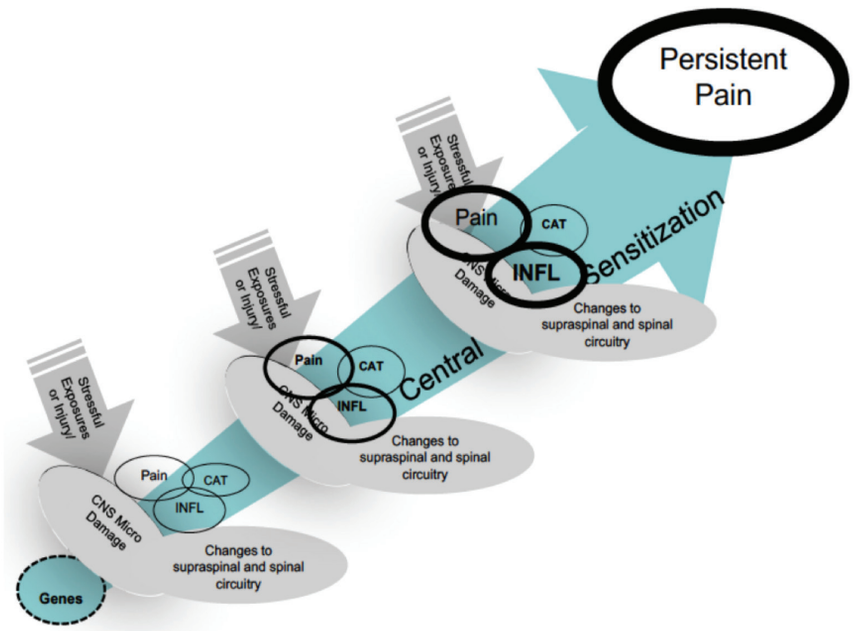

Fig. 1. Developmental model characterising a proposed trajectory of changes in pain sensitivity occurring throughout the lifespan that are related to pain-related catastrophising (CAT). Extensive literature is documenting the changes to the central nervous system (CNS) in response to early childhood exposures to stress and pain. These early changes lay the foundation for later sensitivity to new pain exposures, and parental and social responses to pain also shape this later pain sensitivity. In adulthood, these and new CNS changes occur, particularly when pain becomes persistent. It is possible that brief inflammatory responses (INFL) to pain also increase in magnitude and duration, also contributing to overall sensitivity to new episodes of pain and the persistence of pain over time.
CAT: catastrophising; CNS: central nervous system; INFL: inflammatory responses.

Social

One of the key conceptual models of pain catastrophising, termed the communal coping model, posits that this cognitive-emotional response to pain serves to engage people from the individual's social environment in facilitating coping with the pain (65). The chronic nature of rheumatic diseases creates a long-lasting relationship between the patient and their healthcare provider, which is likely influenced by this cognitive-emotional factor that can be expressed in many ways. Catastrophising can significantly influence the dynamics of the patient-provider interaction and has been shown to predict patient satisfaction with this interaction (66). In turn, the quality of the patient-provider relationship can significantly impact the benefits patients experience from their treatments (67). Of relevance to providers, catastrophising has been recently shown to be associated with using more words expressing sadness and anger in written descriptions of pain (68).

This social dimension to pain catastrophising is particularly relevant to parents managing pain in their children. There is a strong relationship between the levels of parental and child catastrophising (69), and the manner in which parents talk about their child's pain is associated with greater symptom complaints, especially in children with high catastrophising (70). In a longitudinal study on pain memories after children's surgery, pre-surgical catastrophising of both parents and children influenced their memories of postsurgical pain (71). For both adults and children, pain-related disability is an important clinical outcome; parents' catastrophising correlates with their adolescents' level of pain and disability (72). Moreover, the relationship between greater protective parental response (*e.g.* parental permission to stay home from school or receive special privileges due to their pain) and greater adolescent disability is explained by the adolescent's level of catastrophising (73).

Conclusion

Decades of work have demonstrated the importance of pain-related catastrophising in explaining individual differences in response to acute and chronic pain. We now know that catastrophising is in part a learned response to pain that is associated with physiological changes that may have long-term consequences. This concept is particularly important to consider in the care of children and young adults with rheumatic conditions that may expose the individual patient to years of potential suffering into adulthood. We still do not fully understand who develops central sensitisation and how this sensitisation develops over time, yet growing evidence points to specific factors such as pain-related catastrophising (and gender) that increase risk for central sensitisation. Although imperfect, we propose a developmental model (presented in Fig. 1) characterising the trajectory of changes in pain sensitivity that may occur over months and years as pain persists with a chronic painful condition or repeated exposure to intermittent pain.

As patients who are high in catastrophising are less likely to have a robust treatment response to many different types of pain treatments, including psychological treatments, it is important to identify these individuals early and intervene appropriately. This may assist in significantly changing the trajectory of their suffering and disability.

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