Pain-Related Disability in Chronic Pain Patients:

Examining the Roles of Pain Intensity, Pain Catastrophizing and Self-Efficacy through a Moderated Mediation Analysis

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Abstract

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Objective: The literature on chronic pain suggests that pain catastrophizing and self-efficacy are important psychological factors involved in chronic pain. The current study aimed to explore the role of pain catastrophizing and self-efficacy in the relationship between pain intensity and pain-related disability. A hypothetical model was proposed in order to investigate whether the association between pain intensity and pain-related disability was mediated by self-efficacy, as well as examining the potential moderator effects of pain catastrophizing on the direct association between pain intensity and pain-related disability, and on the possible mediation. **Methods:** Analyses were based on a sample of 3.739 outpatients from the Oslo University Hospital Pain Registry (OPR). The OPR is a comprehensive collection of self-reported data on pain characteristics, pain management and physical and mental health. A cross-sectional study was conducted using Baron & Kenny's method of mediation and moderation to examine a simple mediation effect and simple moderation effects. Furthermore, Hayes' conditional process analysis was applied in order to examine a possible moderated mediation effect (conditional indirect effect). The measures included a modified version of the Oswestry Disability Index to assess pain-related disability, a 0-10 Numeric Rating Scale to assess usual pain intensity, The General Self-Efficacy Scale to assess perceived self-efficacy, and The Pain Catastrophizing Scale to assess pain catastrophizing and negative orientation towards pain stimuli. **Results:** The results gave no indication of a moderated mediation effect nor any moderator effects. The simple mediation analysis revealed that self-efficacy partly mediates the association between pain intensity and pain-related disability. **Conclusions:** In accordance with previous research, the results indicated that self-efficacy partly mediates the association between pain intensity and pain-related disability. On the other hand, the results were in disfavor of pain catastrophizing operating as a moderator as proposed in the hypothesized model. The results could imply that there is a less complex association between pain intensity and pain-related disability than the one postulated in our hypothesized model, or that the examined variables relate to each other in a different way than what we postulated.

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1. Introduction

Pain can be defined as a subjective and multidimensional psychological experience. Multidimensionality implies that pain is a result of several perceptual processes and levels of analysis that each have the potential to contribute to the sensation and experience of pain. These processes and levels of analysis can be both psychological and purely physiological phenomena (Hadjistavropoulos & Craig, 2004). When discussed as a psychological phenomenon, pain is commonly described as including sensory-discriminative, cognitive-evaluative, and affective-motivational components. These three general categories encompass several different processes such as nociception, attention, learning, memory, expectation, personality and cultural beliefs. The fact that the experience of pain is a result of a complex combination of factors suggests that pain is constructed in the brain, rather than being an experience that is purely transmitted to the brain (Reddan & Wager, 2019).

A distinction is made between "acute" and "chronic" pain. The term acute pain refers to short-term pain usually caused by tissue damage whereas chronic pain is a persistent experience of pain despite physically adequate healing of the damaged tissue (Hadjistavropoulos & Craig, 2004). Acute pain is an important warning system that indicates danger and is in this regard considered beneficial for survival and health. By definition, all forms of chronic pain have at some point been acute, but somehow failed to cease, ultimately leading to a withstanding experience of pain that no longer serves the originally beneficial function (Badiola, 2016). The transition from acute to chronic pain is, however, not fully understood. Following on the fact that pain is thought to be both a heterogenous and unique experience it appears likely that the transition from acute to chronic pain is equally heterogenous and unique. More specifically, whether or not pain transitions from acute to chronic, and the specific extent of chronicity, is likely to be dependent on subjective human processing.

According to a recent report by the Norwegian Institute of Public Health (NIPH) approximately 30 percent of the Norwegian population report living with chronic pain. NIPH suggests that pain is likely to be the most common reason behind patients consulting their general practitioner. Furthermore, NIPH report that muscular and skeletal disorders are the main diagnosis in 35-45 percent of sick leaves certified by doctors in Norway (NIPH, 2019), as well as being the registered diagnosis in 29 percent of disability benefit cases in Norway (NAV as cited in NIPH, 2019). Analysis of the societal cost of chronic pain has not been carried out in Norway, nor has it been estimated. A thorough analysis in the USA concluded

that the societal costs of chronic pain is equivalent to at least 4.3 percent of gross domestic product annually (IOM as cited in NIPH, 2019). According to NIPH (2019) this percentage would correspond to a cost of at least NOK 135 billion annually. Evidently, chronic pain composes a massive financial cost to the society, in addition to increasing the personal risk of mortality and suicide. Considering that chronic pain causes challenges for public health in addition to personal physical and mental health, it should be regarded as necessary to expand our knowledge on causes, preventative measures and treatment options.

The following article seeks to explore the relationship between pain intensity and pain-related outcomes among patients with chronic pain and explain how psychological factors may influence pain-related disability among chronic pain patients. We propose a hypothesized model consisting of pain catastrophizing and self-efficacy as cognitive components that contribute to the appraisal of pain and thereby the correlation between pain intensity and pain-related disability among chronic pain patients. The overall aim in the field of chronic pain – applying knowledge about mechanisms and causes to determine effective preventative measure and treatments – does however reach beyond the scope of this article.

Nociception, acute pain and chronic pain

Understanding pain as a psychological phenomenon requires a clear understanding of not only the distinction between nociception and pain, but also how pain and nociception relates to one another. Nociception refers to the neurophysiological processing of events that stimulate nociceptors thereby giving rise to a potential pain experience. Pain, on the other hand, is not purely physiological. From the psychological level of analysis, pain is to be understood as a perceptual process involving conscious awareness, selective abstraction, ascribed meaning, appraisal and learning (Hadjistavropoulos & Craig, 2004). In other words, the actual experience of pain is largely dependent on a series of complex cognitive processes including ones that relate to personal beliefs, emotion and motivation. By that logic, nociception and pain respectively largely represents the afferent processes and efferent processes that underlie the experience of pain. The activation of nociception and nociceptive pathways gives an undeniable potential for pain, and several experimental approaches points towards a possible close correspondence between nociception and human pain perception (Hadjistavropoulos & Craig, 2004). There is, however, also withstanding evidence that suggests nociceptive activation can occur in the absence of pain perception. Such findings may possibly indicate that nociception occurring in absence of pain relies on previously established habitual

responses, whereas evoked pain has the potential to produce novel peripheral or spinal cord nociception learning or sensitization (Baliki & Apkarian, 2015). Pain is in this sense to be understood, not as a primitive sensory message of (potential) tissue damage, but as an emergent product of complex wide-spread activity within the brain (Chapman, 2004).

In the field of pain, the terms “acute” and “chronic” represents theoretically and physically distinguishable sensations of pain. Acute pain is an instant and transient type of pain triggered by tissue damage, as opposed to chronic pain, which is more of a permanent sensation of pain that usually exists in the absence of physical tissue damage. Unlike acute pain, chronic pain is usually not adequately explained by physical examination or diagnostic tests and is less receptive to alleviation by conventional medical treatment. Chronic pain typically involves non-noxious stimuli giving rise to a sensation of pain, with the perception of pain being more or less modified by emotional status and other higher cognitive functions such as previous experiences, beliefs and expectation (Seth & de Gray, 2016).

It is postulated that all forms of chronic pain have transitioned from acute pain, although the specific mechanisms behind this transition remains uncertain (Badiola, 2016). As mentioned, pain is a product of wide-spread brain activity and is in this sense involved in the construction of a conscious pain experience. If one were to follow the aforementioned idea, one might also be able to argue that chronic pain represents a faulty learning process where instead of habituating to nociceptive influences the brain simply learns to preserve the sensation of pain. Furthermore, there is reason to believe that some types of emotional distress contribute to the manifestation and continuation of these learnt pain associations (Mansour, Farmer, Baliki & Apkarian, 2014).

General perspectives on the genesis and maintenance of chronic pain

The theoretical understanding of chronic pain has changed considerably over the past four decades. The biomedical model of pain assumed that pain had either a physiological or psychological origin. Any pain response disproportionate to the degree of actual tissue damage would be considered unreal or purely psychological (Turk, Wilson & Swanson, 2010). That is, pain reports unvalidated by objective pathology (tissue damage), gave rise to the assumption that psychological processes were the underlying cause of pain. In instances where objective pathology was identified, other reported symptoms (e.g. hypervigilance to somatic sensations) were considered as secondary reactions of injury or disease, of no importance to physiological pain (Asmundson & Wright, 2004; Turk et al., 2010). A

disadvantage of the biomedical approach to chronic pain is that it does not consider psychosocial factors (Crombez, Eccleston, Van Damme, Vlaeyen & Karoly, 2012). A biomedical approach to chronic pain has been shown to not be enough to understand and treat different problems in daily life that patients experience (Turk, 2005).

As an alternative to the inflexible biomedical model of pain, the biopsychosocial model of illness was introduced to highlight the contributions of complex interactions between biological, psychological and social factors (Engel, 1977; Seth & de Gray, 2016). This model differentiates between objective disease and subjective illness, where objective disease is defined as injury or disruption of body structures or organ systems caused by physiological changes. In contrast, subjective illness is to be understood as the subjective experience of disability or disease (e.g. pain experience), including how the individual, their family and social network respond to and live with the disability (Turk et al., 2010; Turk, Fillingim, Ohrbach, & Patel, 2016).

Subjective illness is viewed as a complex interaction between biological, psychological, social and contextual factors. A biopsychosocial approach to chronic pain presumed that some type of physical pathology is involved, like neurophysiological changes in the joints, muscles or nerves. Moreover, sensory input is transmitted along pathways in the central nervous system through nociceptive fibers to the brain. The sensory information may be interpreted as pain by higher-order psychological processing, involving integration of the individual's learning history, emotional factors and appraisal. This happens within a socio-environmental context, which are all important aspects of pain perception. Psychological factors such as cognitive appraisal is considered as an essential aspect of understanding pain. Appraisal processes involve attributing meaning to somatosensory experiences, expectations of consequences of pain, beliefs about coping strategies and previous learning history (Turk et al., 2016). As such, this model offers a comprehensive framework for understanding chronic pain and the interaction between psychological and non-psychological factors.

Further down the line, Melzack (1999) expanded on this idea by introducing a stress component into the pain equation, ultimately creating The Neuromatrix Model of Pain. According to this model, each individual has a unique neuromatrix constructed by genetics, sensory modalities and cognitive events such as psychological stress. Each individual's personal neuromatrix is thought to play a crucial and equally important part as neural mechanisms and sensory transmissions in determining the overall interpretation of the

experience of pain (Melzack, 1999). Biological factors, including gender, genetics and underlying diseases, may be involved in the initiation of nociception as well as maintenance and regulation of the pathophysiological changes in pain (Seth & de Gray, 2016). A rule of thumb is that psychological variables affect how one evaluates and perceives the pain experience, whereas social factors affect behavioral responses to pain (Seth & de Gray, 2016). Although important concerns in relation to pain, this paper is concerned primarily on the psychological factors relating to the genesis and experience of chronic pain, and as such biological and social factors will not be further discussed in isolation.

The fear-avoidance model

Several specific models framed within the biopsychosocial perspective aim to describe the processes that influence pain-related outcomes. The fear-avoidance model is a widely known model, and was originally formulated to explain the development and persistence of low back pain, but has later been expanded on (Crombez et al., 2012; Edwards, Dworkin, Sullivan, Turk & Wasan, 2016; Lethem, Slade, Troup & Bentley, 1983; Vlaeyen, Kole-Snijders, Boeren & Eek, 1995). The model describes pain-related disability as a result of interaction between fear-related cognitive, affective and behavioral processes (Zale & Ditre, 2015). Fear of pain is described as the central component of the model; pain can either be interpreted as non-threatening or threatening by the individual. Pain interpreted as non-threatening involves perceiving pain as something temporary, where individuals return to daily activities after a short period of inactivity. Interpreting pain as threatening involves pain to be wrongly interpreted as a serious injury (Crombez et al., 2012: Lethem et al., 1983; Vlaeyen et al., 1995). Pain catastrophizing is hypothesized as a key factor in the model, describing that individuals with a trait tendency to catastrophize are predisposed to develop and maintain chronic pain compared to patients without this tendency. This is related to evidence indicating that individuals with a tendency to catastrophize are inclined to display an exaggerated reaction to both actual and potential threats, and to some extent hyperfocus on pain, which ultimately may result in greater pain intensity (Martinez-Calderon, Jensen, Morales-Asencio & Luque-Suarez, 2019; Vlaeyen et al., 1995). Attentional processes such as hypervigilance to pain were not initially included in the original model (Crombez et al., 2012; Lethem et al., 1983), but it emphasizes an orientation towards pain-related information at the expense of non-pain-related information, thereby demonstrating mechanisms in maintenance and development of chronic pain (Crombez et al., 2012; Crombez, Van Damme, & Eccleston, 2005).

The fear-avoidance model hypothesizes that pain catastrophizing can lead to excessive fear of pain and further decrease physical activity by encouraging avoidance of movements and activities the individual has learned to associate with pain (Martinez-Calderon et al., 2019; Vlaeyen et al., 1995). Avoidance is likely to impede the process of adjusting expectations to actual experiences, which may result an in overestimation of the negative effects of pain as well as aggravation of physical and mental health symptoms. This pattern of behavior could potentially result in increased vulnerability to pain in the future (Crombez et al., 2012). Evidence has consistently indicated that pain-related fear is positively associated with disability, and that it may be an essential factor in predicting the continuation of disability (Zale & Ditre, 2015). Moreover, pain catastrophizing seems to predict both pain intensity and pain-related disability (Severeijns, Vlaeyen, van den Hout & Weber, 2001). However, some research has generated results disconfirming that changes in pain catastrophizing is associated with changes in pain intensity, while still supporting the hypothesis of an association to pain-related disability (Woby, Watson, Roach & Urmston, 2004). The inconclusiveness suggests some uncertainty regarding the specific relationship between pain catastrophizing and pain intensity.

The fear-avoidance model has been extended by different authors to include posttraumatic stress (Turk, 2002), and the extended understanding of pain catastrophizing being associated with certain dispositions such as anxiety sensitivity and trait anxiety (Crombez et al., 2012; Sullivan, Thorn, Rodgers & Ward, 2004). A further extension of the model is suggested by Crombez et al. (2012), claiming that there remain some issues to be explored. They argue that the model fails to address how individuals try to function despite pain and recovery processes, remarking that the fear-avoidance model does not offer a satisfactory explanation of how the process of gradually resuming to daily activities leads to recovery. A further challenge related to the motivational aspect of pain is how the goal of avoiding or controlling pain competes with other goals in everyday life. It is hypothesized that pain is more than an emotional reaction associated with harm, it is a disruptive experience that prevents individuals from achieving valued goals (Crombez et al., 2012).

Crombez et al. (2012) presents an integration of a motivational perspective in the fear-avoidance model. In the context of chronic pain, goal persistence may refer to the ability to ignore pain and withstand in order to accomplish a set goal. There is some evidence of associations between endurance behaviors, such as thought suppression and perseverance, and pain-related disability. Although there is a lack of firm evidence, there is some indication that

endurance behaviors despite pain may or may not become dysfunctional in the long term, depending on if the endurance behavior is combined with task persistence or avoidant behavior (Crombez et al., 2012; Hasenbring & Verbunt, 2010). Another suggestion by Crombez et al. (2012) for an extension to the fear-avoidance model is that individuals with chronic pain may begin to focus on achieving pain relief itself rather than their current and valued goals in daily life. This is proposed to be an unproductive problem-solving strategy for chronic pain patients. In the acute stage of pain, pain catastrophizing and avoidance of potentially injurious movements might reflect an adaptive attempt at problem-solving because the individual try to resolve the issue. However, if no solution is obtainable this may become dysfunctional for chronic pain patients. Additionally, learnt responses might explain avoidance of pain-inducing situations if the individuals have repeatedly experienced that pain interferes with attaining desired goals (Crombez et al., 2012).

In the context of recovery, Crombez et al. (2012) refers to theories of self-regulation and suggest two approaches that might facilitate recovery; goal disengagement defined as minimizing the effort to attain unattainable goals, and goal reengagement defined as establishing and focusing on alternative goals. Evidence indicates that adjusting unrealistic goals serve as a protective factor against the disadvantageous effects of goal failure, and also shown to increase quality of life (Wrosch, Miler, Scheier, & Brun de Pontet, 2007). Furthermore, acceptance is suggested to facilitate recovery, in terms of refocusing attention from pain relief to rewarding and positive daily life activities (Crombez et al., 2012). In summary, the fear-avoidance model is a useful model in understanding chronic pain and disability, but further research concerning a motivational perspective on chronic pain is essential.

The transactional model of stress

In pain literature several theoretical perspectives have been proposed. From a cognitive-behavioral perspective, the experience of chronic pain is suggested to be related to behavioral, affective, cognitive and sensory factors, though the individual's beliefs and perceptions associated to the pain experience is emphasized (Novy, Nelson, Francis & Turk, 1995). A model to first incorporate both cognitive and behavioral components is the biobehavioral model where a diathesis-stress interaction is suggested, combining biological, psychological and sociocultural factors (Turk, 2002).

Even though there are several relevant models within the cognitive-behavioral perspective, the transactional model of stress is of particular interest (Lazarus & Folkman, 1984). The model suggests a theoretical framework in understanding adjustment and adaptation to chronic pain (Ramírez-Maestre, Esteve & López, 2008). The model defines psychological distress as "a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being" (Lazarus & Folkman, 1984, p. 19). Researchers argue that chronic pain can be viewed as a stressor since it is a threatening experience and interferes with tasks in everyday life (Thorn, 2017; Van Damme, Crombez & Eccleston, 2008).

A conceptualization of chronic pain within the transactional model of stress has been proposed by Thorn (2017), with an emphasis on cognitive factors. Lazarus & Folkman (1984) argue that personality, social and biological factors all play an important role in how individuals interact, responds and cope with a stressor, including appraisal processes. According to the transactional model of stress, appraisal processes are defined as cognitions involving evaluative processes of situations or stimuli (Thorn, 2017; Lazarus & Folkman, 1984).

The transactional model of stress proposes two distinguishable appraisal processes, primary appraisals and secondary appraisals. Primary appraisal is defined as judgments about whether a potential stressor is irrelevant (the stimuli does not interfere with the individual's well-being), benign-positive (the stimuli enhance well-being), or stressful. Stress appraisals include three distinctive constructs: harm/loss, threat and challenge appraisals. Harm/loss refers to actual physical and psychological damage (Lazarus & Folkman, 1984). An example relevant for chronic pain patients is that they report loss of income, difficulties in relationships and decreased activity levels compared to the period before developing a chronic pain condition (Thorn, 2017). Threat appraisals involve perceptions of potential harm or losses and its implications of well-being. The third stress appraisal, challenge appraisals, involves the perception that the individual's ability or resources to cope are not exceeded, thus the individual is able to focus on the potential growth in relation to the stressor (Lazarus & Folkman, 1984; Ramírez-Maestre et al., 2008). Furthermore, both threat and challenge appraisals can occur concurrently since the presence of one of them do not necessarily exclude the other (Lazarus & Folkman, 1984).

Secondary appraisals involve evaluation or beliefs in an individual of what can be done when facing danger or a stressor, or in other words the evaluation of effectiveness of coping responses. Like primary appraisals, secondary appraisals can elicit emotions and influence one's choice of coping options (Lazarus & Folkman, 1984). According to Thorn (2017), pain is considered a stressor when an individual perceives that the pain experience surpasses his or her resources or ability to cope with the pain. Furthermore, Thorn (2017) argues that automatic thoughts and acquired beliefs about the pain condition can be understood as secondary cognitive appraisal processes. Automatic thoughts seem to some extent operate on a preconscious level, arising spontaneously in response to a stimulus. Acquired beliefs include assumptions about the self, the world or a situation, pain beliefs or one's ability to cope with pain. Automatic thoughts and acquired beliefs are both involved in self-efficacy and pain catastrophizing and can be understood as secondary appraisals. However, when it comes to pain catastrophizing, it has been conceptualized as both a primary and secondary appraisal and as a coping strategy in the pain literature (Thorn, 2017). In other words, a lacking consensus regarding the conceptualization of pain catastrophizing is evident (Jackson, Wang & Fan, 2014a).

Pain coping classification

The concepts of coping and defenses include both adaptive and maladaptive habitual styles in the management of experiences that pose a threat to emotional equilibrium (Cramer, 1998; Malone et al., 2013). Some have hypothesized that defense mechanisms represent a psychopathological form of managing, while claiming that coping mechanisms are part of normal psychological functioning. The Freudian definition does, however, clearly state that defenses are part of normal functioning and development provided that one is able to refrain from excessive and inappropriate application (Cramer, 1998). Furthermore, coping is not inherently adaptive, and some studies have in fact found a more frequent association between select coping scales and negative outcomes rather than positive outcomes (Cramer, 1998). It seems then that the normality/pathology dimension represents a faulty distinction between defense and coping, suggesting that both coping mechanisms and defense mechanisms are relevant factors in the context of psychological adjustment and maladjustment. In the context of pain, these processes are described as initial appraisals of pain and evaluation of available resources to manage pain. Based on these appraisals, some cognitive, emotional or behavioral response is made to cope with the pain (Tan, Teo, Anderson & Jensen, 2011).

Several coping classification systems have been proposed. Pain coping has frequently been classified as active and passive responses, the former referring to a response involving an attempt to function despite pain or manage pain by utilizing personally available resources, whereas the latter refers to any response characterized by helplessness, withdrawal or surrendering control over pain (Snow-Turek, Norris & Tan, 1996). When classified as adaptive and maladaptive, pain responses are organized by the expected effects of the coping response. Although they are separate approaches to classification, the coping responses classified into the respective categories tend to be similar across classification systems. Specifically, active responses are in large found to be more adaptive, and passive responses are typically viewed as maladaptive (Tan et al., 2011). Earlier research has demonstrated a consistent association between certain psychosocial factors and functioning and adjustment in individuals with primary chronic pain problems. Among these factors are pain-related catastrophizing cognitions, pain related beliefs and attributions as well as social and environmental variables (Osborne, Jensen, Ehde, Hanley & Craft, 2007).

It has been hypothesized that negative appraisals of pain related problems render patients with an incline to mobilize passive coping strategies during daily activities, ultimately leading to reduced activity levels and functioning. On the other hand, it has also been hypothesized that challenge appraisals predict the practice of active coping strategies. Specifically, a study demonstrated an association between high levels of challenge appraisals and low levels of passive coping. By contrast, evidence indicate that harm, loss and threat appraisal is associated with heavier application of passive coping strategies, which in turn was associated with higher level of pain-related disability as well as higher levels of pain intensity (Ramírez-Maestre et al., 2008). In terms of self-efficacy, a study involving a sample of Chinese low back pain patients indicated that self-efficacy was indirectly positively related to quality of life through active coping (Du et al., 2018).

As previously mentioned, the current body of literature lacks a clear consensus on whether pain catastrophizing can be understood as a primary or secondary appraisal, or as a coping strategy (Jackson et al., 2014a). Some researchers suggest that pain catastrophizing functions as an avoidant (passive) coping strategy because of a disadvantageous emphasis on abstract negative cognitions, which prevents an individual from engaging in more constructive problem solving (Flink, Boersma & Linton, 2013). This could possibly explain the multiple observations of an association between pain catastrophizing with negative pain-related outcomes (Martinez-Calderon et al., 2019).

In sum however, the available evidence indicates both beneficial and adverse effects on pain from several coping strategies, hence no coping strategy occurs to be unambiguously more effective than others. The evaluation of a coping strategy's efficacy is hinged on context, i.e. one specific coping strategy might be passive in one situation yet considered as active and problem focused in another (Van Damme et al., 2008). When further relating this to the leading theoretical perspectives within the field of chronic pain, it seems that which adaptive and maladaptive behaviors that contribute to the development and maintenance of chronic pain might not be found in a specific strategy. Rather the answer might be found within a more general, broader and perhaps emotional factor due to their influences on the process of selecting a coping strategy and therefore also on observable behavior.

Self-efficacy and chronic pain

Perceived self-efficacy refers to a psychological mechanism concerned with an individual's subjective evaluation of his or her own capability to successfully complete a given task or their ability to effectively control a situation (Bandura, O'Leary, Taylor, Gauthier & Gossard, 1987). Bandura et al. (1987) argued that judgements of self-efficacy influence the choice of action, the amount of effort put into an attempt, perseverance during aversive experiences, whether thoughts benefit or interrupt an attempt, and the amount of stress experienced in a demanding situation. Keeping this in mind, it is further argued that perceived coping efficacy might facilitate pain relief. The belief in a personal ability to alleviate or avoid suffering is likely to mobilize skills that are relevant for alleviating pain and provide the needed motivation to persevere, all the while reducing aversive anticipations that has the potential to intensify pain and discomfort (Bandura et al., 1987). In terms of the transactional model of stress, Thorn (2017) argues that self-efficacy can be understood as a secondary appraisal.

Research by Soares & Grossi (2000) demonstrated a negative correlation between anxiety/depression and subjective self-evaluations of performance potential (here: termed as self-esteem). This finding might indicate that patients with low levels of anxiety/depression simultaneously display a higher subjective evaluation of self-efficacy, thus suggesting that the level of self-efficacy might be contingent on the intensity of symptoms of anxiety and depression. In addition, their study also showed a positive association between pain intensity and self-esteem when controlling for anxiety/depression, thereby suggesting that this specific relationship is influenced by the presence of anxiety and depression symptoms (Soares & Grossi, 2000). They proposed that a high level of self-esteem serves as a protectant against

emotional distress and facilitates coping with intense pain, as supported by the positive association between self-esteem and active coping (Soares & Grossi, 2000). Furthermore, others have found associations between self-efficacy and resilience, proposing that higher resilience is positively correlated with greater pain self-efficacy (Newton-John, Mason & Hunter, 2014; Stewart & Yuen, 2011). Resilience has been defined as a capacity of being successful in maintaining or regaining mental health facing danger or adversity. It focuses on protective psychological factors, rather than on risk factors and psychopathology (Stewart & Yuen, 2011). Resilience is known to be relevant to various clinical populations and likely applies to individuals with chronic pain as well (Newton-John et al., 2014).

Generally, self-efficacy has been described as a protective psychological resource in chronic pain patients. A meta-analysis evaluated the relationship between self-efficacy and functioning (e.g. functional impairment, pain severity, affective distress) in patients with chronic pain, and found negative correlations between self-efficacy and impairment, pain severity and affective distress, arguing that self-efficacy serves as a protective factor among chronic pain patients (Jackson, Wang, Wang & Fan, 2014b). Evidence also suggests that higher ratings of self-efficacy is associated with less catastrophizing, reduced avoidance of pain, less disability, less pain severity and depression (Perry & Francis, 2013).

In a longitudinal study of a population consisting of patients with low back pain, it was found that changes in self-efficacy beliefs at 12 months after onset of chronic back pain mediated the relationship between changes in pain and changes in disability, whereas fear of movement beliefs did not mediate this relationship. A clinical implication from this study suggests that increasing self-efficacy rather than only targeting reductions in pain intensity could be beneficial. The researchers suggest that it could be relevant in clinical practice to identify patients with low levels of self-efficacy, expecting these patients to benefit from psychological interventions to enhance self-efficacy, in addition to medical interventions (Costa, Maher, McAuley, Hancock & Smeets, 2011). Additionally, previous research supports the role of self-efficacy operating as a mediator between pain intensity and pain-related disability in chronic pain patients with a variety of medical diagnoses and pain locations such as lower back, head/neck, extremities, chest, abdomen or pelvis (Arnstein, 2000; Arnstein, Caudill, Mandle, Norris & Beasley, 1999). Furthermore, a study including a Japanese outpatient group with various types of chronic pain conditions found that increase in self-efficacy was associated with reduction in disability. This supports the clinical implication to

improve self-efficacy to reduce disability among chronic pain patients (Karasawa et al., 2019).

Chronic pain, pain catastrophizing and pain learning

Pain catastrophizing has been defined as a negative cognitive and emotional orientation to pain commonly characterized by three highly correlated components – rumination, magnification and helplessness toward actual or anticipated pain (Feinstein et al. 2017; Sullivan, Bishop & Pivik, 1995). It is a widely studied cognitive variable thought to be one of the most powerful influences on pain perception and pain-adjustment, and thereby one of the most powerful predictors of poor outcomes in chronic pain samples (Feinstein et al. 2017; Martinez-Calderon et al., 2019; Thorn, 2017).

Rumination refers to a cognitive process involving the tendency to maintain emphasis on pain and its impact. Magnification represents a tendency to exaggerate the severity and threat of pain, while helplessness represents a belief that one is unable to cope with the pain. Sullivan et al. (1995) argued that magnification and rumination may be related to primary appraisals in which individuals exaggerate the threat value of pain stimuli, and that helplessness may be related to secondary appraisal processes in which individuals negatively evaluate their ability to cope effectively with pain. A meta-analysis found a positive correlation between threat appraisals and increased pain intensity, impaired functioning, use of passive coping strategies, avoidance of activities associated with pain and emotional stress. However, it is important to note that measures of pain catastrophizing in these studies was included as a passive coping strategy, highlighting the ongoing debate on the conceptualization of pain catastrophizing (Jackson et al., 2014a).

From a cognitive-behavioral perspective, pain catastrophizing can be viewed as a maladaptive cognitive response. Fear in response to acute injury is viewed as adaptive as the individual can limit physical activity to promote healing. One the other hand, increased attention to pain could possibly create an exaggerated tendency to catastrophize about potentially painful stimuli, especially in the context of a small or no risk for physical damage, which may result in an establishment of disadvantageous pattern of cognitive and behavioral inhibition (Quartana, Campbell & Edwards, 2009). Avoidance of situations or movements that can elicit pain is also a central predictor of pain-related disability, where the individual not only avoids pain but also experiences negative emotions related to anticipation of pain (Van Damme et al., 2008).

A recent meta-analysis found that higher levels of pain catastrophizing in individuals with chronic musculoskeletal pain was associated with and predicted both chronic pain intensity and disability (Martinez-Calderon et al, 2019). Evidence also indicates that greater catastrophizing was associated with greater pain intensity, and that catastrophizing is a significant predictor of pain-related disability in chronic pain (Arnow, Blasey, Constantino, Robinson & Hunkeler, 2011; Turner, Jensen, Warms & Cardenas, 2002). Additionally, previous research reported that pain catastrophizing operated as a moderator between daily psychical activity and pain intensity in patients suffering from knee pain (Lazaridou et al., 2019).

It has been proposed that exposure to painful situations or catastrophic reactions to pain in other individuals gives rise to a learning context wherein one is made to associate high threat of painful stimuli with an inability to effectively cope with pain. This type of association may contribute to development of adverse cognitive schemas on pain coping and pain management (Sullivan, et al., 2001; Quartana et al., 2009). The sensation of pain in the absence of nociceptive pathways, as seen in patients with phantom pain, strongly indicates that pain is in fact partly a psychological phenomenon, which furthermore implies the likeliness of learning through both cognitive and emotional appraisal (Mansour et al., 2014). Apkarian (2008) proposed the following definition of the relationship between pain, learning and memory: "Chronic pain is a persistence of the memory of pain and/or the inability to extinguish the memory of pain evoked by an initial inciting injury" (Apkarian, 2008, p. 466). This viewpoint is, from an evolutionary perspective sensible, as the survival value of pain is contingent on an individual's ability to learn and remember. Following on this, it has been hypothesized that chronic pain reflects a state of continuous learning wherein aversive emotional associations are consecutively formed within incidental events simply because of a persistent presence of pain. Simultaneously, continued presence of pain reduces any possibility of extinction due to the subject continuously being re-exposed to the conditioned event whilst still in pain, thereby reinforcing the aversive association (Apkarian, 2008).

Adaptive and maladaptive responses to pain: the cognitive connectors

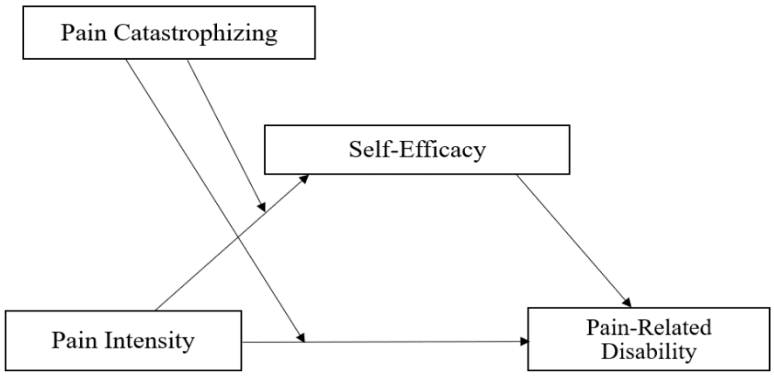
The main takeaway provided by the research literature appears to be that the development and maintenance of chronic pain is complex and multifaceted, including factors relating to physiology, psychology, environment and social context. Two factors do however appear to stand out as key psychological factors with substantial importance regarding the transition to

chronic pain as well as the maintenance of chronic pain, mainly due to their overall potentially debilitating impact. Based on this knowledge, we suggest that pain catastrophizing and self-efficacy may be understood as some executive or higher-order factors highly significant for the overall appearance, display and trajectory of chronic pain. Despite the literature being inconclusive regarding the effectiveness of several specific coping strategies, current evidence seems to point towards a connection between self-efficacy and the appearance and characteristics of chronic pain. Specifically, the degree of self-efficacy seems to be of great importance for the possibility to select advantageous strategies to reduce the experience of pain, as suggested by several reports of self-efficacy mediating the association between pain intensity and pain-related disability (Arnstein et al., 1999; Arnstein, 2000; Costa et al. 2011). Moreover, pain catastrophizing appears to emphasize attention to pain sensations, increasing the likelihood of the individual selecting disadvantageous coping methods. For instance, Severeijns et al., (2001) found evidence that chronic pain patients with a greater tendency to catastrophize experience more pain intensity, report more pain-related disability as well as more psychological distress. The same study also concluded that pain catastrophizing is a critical predictor of disability.

With that in mind, we hypothesize a relationship between pain intensity and pain-related disability influenced by two cognitive connectors: self-efficacy and pain catastrophizing. More specifically, we hypothesize a relationship where the association between pain intensity and pain-related disability is mediated by self-efficacy and moderated by pain catastrophizing, as depicted in Figure 1.

Figure 1

A hypothesized model of how pain intensity and pain-related disability may be associated with pain catastrophizing and self-efficacy



As our hypothesized model suggests, pain intensity might predict pain-related disability through both an indirect and direct effect. A possible indirect effect can be seen on the upper part on the model, with self-efficacy acting as a mediator between pain intensity and pain-related disability. Specifically, the degree of pain intensity is presumed to influence perceived self-efficacy. Furthermore, this assumed relationship could be dependent on the degree of pain catastrophizing. Based on these assumptions we hypothesize that a higher level of pain intensity combined with a greater tendency to pain catastrophize is associated with lower self-efficacy, which in turn results in greater pain-related disability. As for the direct effect between pain intensity and pain-related disability, pain catastrophizing acts as a moderator, interacting with pain intensity to determine pain-related disability. The model then suggests two independent interactions, with pain catastrophizing influencing both the direct effect and indirect effect.

Four separate hypotheses derive from our hypothesized model: (1) there will be a positive correlation between pain intensity and pain-related disability, (2) self-efficacy mediates the association between pain intensity and pain-related disability, (3) pain catastrophizing moderates the association between pain intensity and pain-related disability, and (4) pain catastrophizing moderates the association between pain intensity and self-efficacy. Following this, we hypothesize that all sub-hypotheses operate in a single model, the hypothesized model depicted in Figure 1.

2. Methods

The OPR questions and questionnaires

The Oslo University Hospital Pain Registry (OPR) is the most comprehensive pain registry for the largest interdisciplinary outpatient pain clinic in Norway, treating both acute and chronic pain conditions (Oslo University Hospital, n.d.; Oslo University Hospital, 2020).

Data registration began in October 2015, and is carried out continuously, with approximately 1,000 patients assessed each year (Granan, Reme, Jacobsen, Stubhaug & Ljoså, 2019). The patients complete an electronic registration upon arrival to their first consultation at the outpatient clinic. The baseline questions of the OPR include basic demographics; age, sex, cohabiting, marital status, number/age of children, education, employment, social benefits, self-rated evaluation of personal economy, application for disability pension and litigation due to pain condition. In addition to demographics, the OPR also includes several questionnaires assessing functional ability, pain location, pain intensity, duration of pain condition, psychological distress, fatigue, insomnia, pain catastrophizing, perceived self-efficacy, and self-rated health/wellbeing.

Sample

The following analyses are based on a sample of 3.739 patients from the OPR. All patients are either referred from a general practitioner or specialist health care services to the pain clinic. Pain conditions include chronic pain with and without a known precipitating cause, pain associated with cancer, heart disease, chronic obstructive pulmonary disease (COPD) and multiple sclerosis and subacute pain conditions with risk of chronification (Norwegian Directorate of Health, 2015). However, pain conditions such as chronic nonmalignant pain is overrepresented (Oslo University Hospital, 2020). Treatment are offered in the local area (Oslo) and South-Eastern Health region in Norway. Following treatments are offered: group therapy, cognitive therapy, physiotherapy, medical treatment, spinal cord stimulation, radio frequency therapy and more. In addition, the pain clinic offers acute treatment to surgical inpatients and inpatients suffering from injury (Oslo University Hospital, n.d.). Although data collection has been continuous since October 2015, our analyses were carried out with data collected in the period from January 2016 to March 2020.

Procedure

All patients attending the pain clinic meet one hour prior to their first scheduled consultation to provide relevant information to the registry. A pre-programmed tablet guides the patients through a series of questions and questionnaires, requiring 20-45 minutes to complete. Patients unable to perform an electronic registration are provided with a paper-based form. A short form of the registry questionnaire package is provided to patients with cognitive impairments or patients who are otherwise prevented from completing the complete package.

Measures

The analyses are based on a selection of completed questionnaires from the OPR including a modified Oswestry Disability Index (ODI) to assess pain-related disability; 0-10 Numeric Rating Scale (NRS) to assess usual pain intensity; the General Self-Efficacy scale (GSE) to assess perceived self-efficacy irrespective of morbidity and disability; and Pain Catastrophizing scale (PCS) to measure pain catastrophizing and a disproportionate negative orientation toward pain stimuli and pain experience.

Questionnaires

Pain-related disability was measured using a modified Oswestry Disability Index. The original ODI is among the most commonly applied scales for outcome measures for individuals with low back pain. It is a self-administrated questionnaire containing 10 items concerning back pain and different activities of daily life, requiring approximately five minutes to complete (Granan, et al. 2019; Vianin, 2008). Each item is scored on a range from 0 to 5, with 5 representing the greatest disability. Scores reflect the patients' degree of disability on a range from minimal to bedbound. The scores are calculated to a percentage score by dividing the obtained total score with the total possible score (50) and multiplying this score by 100 (Fairbank & Pynsent, 2000). Research has shown the original ODI to be valid, reliable and responsive (Granan et al., 2019; Vianin, 2008). The ODI is validated in Norwegian, showing high construct validity and internal consistency of .94 (Grotle, Brox & Vøllestad, 2003). The modified ODI used in the OPR is identical to the original ODI apart from the word "back" being removed. The modified ODI is thus suitable to measure functional outcome among a variety of pain conditions (Granan et al., 2019).

Pain intensity was measured by NRS. The NRS is an 11-point numerical scale ranging from 0 to 10, respectively from no pain to worst possible pain (Williamson & Hoggart, 2005). The

numeric rating scale is considered to be as reliable and valid as other pain intensity rating scales (e.g. Visual Analogue Scale, Faces Pain Scale-Revised and Verbal Rating Scale). It is also the preferred form of measurement due to the simple administration and scoring, as well as being sensitive to differences in pain intensity over time (Ferreia-Valente, Pais-Ribeiro & Jensen, 2011; Jensen, Karoly & Braver, 1986; Williamson & Hoggart, 2005).

A Norwegian version of the General Perceived Self-Efficacy Scale (GSE) was included as a measure of the patient's perceived self-efficacy irrespective of morbidity and disability. The scale contains a total of 10 items concerning an individual's optimistic self-belief in the personal ability to cope with a variety of stressful or challenging demands. The possible responses range from 1-4; *not at all true (1), hardly true (2), moderately true (3),* and *exactly true (4).* The Norwegian version of the GSE was translated by Espen Røysamb through the translation back-translation procedure. In their study of the psychometric properties and validity of the GSE, Leganger, Kraft & Røysamb (2000) confirmed the GSE to be a valid and reliable measure of perceived self-efficacy, reporting internal consistency of $\alpha = 0.82$, and a Guttman split-half reliability coefficient of .78. Additionally, construct validity was also found to be satisfactory (Leganger et al., 2000).

The Pain Catastrophizing scale (PCS) measures pain catastrophizing and negative orientation toward pain stimuli and pain experience. The PCS is a 13-item instrument, in which each item is scored on a 5-point scale ranging from *not at all (0), to a slight degree (1), to a moderate degree (2), to a great degree (3),* and, *all the time (4).* Scoring yields a total score as well as three subscale scores; rumination, magnification and helplessness (Sullivan et al., 1995). Research has documented high internal consistency and reliability of the full-scale score, with Cronbach's α ranging from .91 to .93. Scores on rumination, magnification and helplessness subscales range from .87 to .93, .70 to .77 and .76 to .87, respectively (Osman et al., 1997). High construct validity is also reported for the PCS (Osman et al., 1997; Sullivan et al., 1995). Research on the psychometric properties of the Norwegian PCS confirms a high internal consistency with a Cronbach's α of .90 for the full-scale score as well as the subscales rumination and helplessness, with a Cronbach's α of .83 and .86, respectively. With a Cronbach's α of 0.53, it is however evident that the magnification subscale lacks satisfactory internal consistency in the Norwegian translation of PCS (Fernandes, Kjersti, Lochting & Grotle, 2012).

Analyses

All analyses were performed in IBM SPSS Statistics 26, with the exception of Sobel test.

General Data Cleaning

A general data cleaning was performed prior to conducting the statistical analyses. This included checking missing values as well as recoding erroneous values. GSE was recoded by reversing the scores to simplify the interpretation of the results. In the recoded variable, low scores represent higher self-efficacy and high scores represent lower self-efficacy. All analyses were performed using listwise deletion. The considerable differences in missing values might attributable to whether each patient has chosen to fill out the complete package of the questionnaires or the short version. The complete package includes all scales, whereas the short version includes ODI, NRS and PCS. One possible reason for the significant difference between GSE and the other measures might be that many patients choose to complete the short version instead of the full version. In addition, missing values for ODI is considerably lower because patients who are unable to the complete the short version are given ODI and one additional scale. Missing data are presented in Table 1.

Table 1

Missing data for the questionnaires ODI, NRS, PCS and GSE

	ODI	NRS	PCS	GSE
N	3737	3358	3424	3064
Missing	2	381	315	675

ODI = Oswestry Disability Index; NRS = 0-10 NRS Usual Pain Intensity; PCS = Pain Catastrophizing Scale; GSE = General Self Efficacy Scale

Preliminary analyses

In order to perform a linear regression analysis (moderated mediation), evidence of potential bias in the data was examined. These assumptions include normality, multicollinearity, homoscedasticity, linearity and presence of outliers. Violations of these assumptions can potentially bias parameter estimates and validity of the results (Field, 2013; Hayes 2018).

Preliminary analyses were carried out using the Linear Regression command with ODI as a dependent variable and PCS, GSE and NRS as independent variables. The normality

assumption was examined with P-p plots which assess whether the residuals for the outcome variable are normally distributed. P-p plots indicated that the data is normally distributed and suggested that the normality assumption was met. Homoscedasticity examine whether the variance of the scores on the outcome variable are equally distributed across the independent variables (Hayes, 2018). The assumption was examined by a scatterplot of the observed residuals against the predicted values of the outcome variable (Field, 2013). The scatterplot revealed that the assumption of homoscedasticity was also met to a satisfactory degree. According to Field (2013), this also reveals that the assumption of linearity is met. Nevertheless, an additional examination of linearity was performed through visually examining scatterplots between ODI and PCS, NRS and GSE. Scatterplots reveals whether a linear or curvilinear relationship is present (Field, 2013). The scatterplots revealed a linear relationship between the variables, with no indications of curvilinear relationships, confirming that this assumption was in fact met.

The presence of outliers was examined. Outliers could severely impact the slope of the regression line and the validity of the results (Bordens & Abbott, 2014). Therefore, the presence of outliers was examined through boxplots of the scales included in this study, and revealed outliers for the scales GSE, ODI and NRS. Cook's distance test was applied to examine the potential influence of the outliers, which assess the influence of a case on the model's ability to predict all the cases. Values greater than 1 indicate a potential concern (Field, 2013). Cook's Distance test revealed no cases with greater value than 1, signifying that the outliers constitute an insignificant amount of influence on the data. However, it is also worth noting that in clinical studies as this one, outliers might not necessarily be a reason for concern as all values represent a natural and valid variability in the scores. Therefore, outliers might not represent an invalid score that would artificially change the results.

Multicollinearity refers to highly correlated independent variables, potentially undermining the statistical significance of the independent variables. Presence of high multicollinearity between the independent variables makes it difficult to extricate the separate effect of each variable, providing little unique information (Bordens & Abbott, 2014; Field, 2013; Hutcheson & Sofroniou, 1999). The correlation matrix revealed an absence of multicollinearity between the independent variables, NRS, the moderator variable PCS and the mediator variable GSE, with correlations of $r > .9$. The assumption of multicollinearity was further examined through VIF and tolerance values. All VIF values were less than 10 (highest VIF-value = 1.32), and tolerance values were above .2 (lowest tolerance value = .75),

indicating no concern regarding multicollinearity. In sum, assumptions for parametric tests were met (Field, 2013).

Statistical analyses

The following statistical analyses were performed: 1) correlational analyses, 2) hierarchical regression analysis to examine mediation effect, followed by Sobel test and Hayes PROCESS, 3) two hierarchical regression analyses to examine moderation effects and 4) moderated mediation analysis (model 8) following Hayes (2018) analytical strategy.

Correlations

The first analysis focused on examining the correlational association between ODI, GSE, NRS and PCS. The correlational analysis was performed to establish whether a significant association between the reports on ODI and NRS was present or not. Furthermore, the correlational analysis was performed to establish whether a significant association between all the variables was present. The analysis was carried out through Pearson's Bivariate correlations.

Hierarchical regression analyses

A hierarchical regression method was applied to examine mediation and moderation effects. In hierarchical regression analysis, several regression models are built to examine whether an independent variable statistically significantly explains variance of the dependent variable after adding it to a previous model. This is examined through presence of a significant improvement in R^2 (explained variance) after adding the new independent variable to the model (Field, 2013; Hajovsky & Reynolds, 2018). A significance level of $p < .05$ was selected.

Mediation effects

In order to perform a moderated mediation analysis, hierarchical regression analysis was performed to examine mediation effect following Baron & Kenny's (1986) method. The concept of mediation represents a hypothesized causal chain wherein a given third variable is suggested to partly or fully account for the relationship between the independent variable and the dependent variable (Baron & Kenny, 1986). According to Baron & Kenny (1986), three general criteria need to be met in a mediational relationship: 1) the independent variable must significantly affect the presumed mediator, 2) the presumed mediator variable must significantly affect the dependent variable, and 3) a previously significant relationship

between the predictor and the criterion is no longer significant when including the presumed mediator variable. A full mediation effect is present in cases where the mediator variable completely explains the correlation between the independent variable and the dependent variable. On the other hand, one would have a partial mediated effect if the independent variable remains partly accountable for the variation in the dependent variable after including the mediator. A partial mediation effect could imply that the variations in the dependent variable is accounted for by several mediating variables.

A simple mediation analysis was considered necessary to confirm the presence of a mediation effect, as an absence of a significant mediation effect would invalidate our hypothesized model. In step 1, ODI was entered as a dependent variable and NRS as an independent variable. In step 2, GSE was entered as a dependent variable, and NRS as an independent variable. In step 3, NRS and GSE were entered as independent variables and ODI as a dependent variable. This was followed up by Sobel test using an online calculation tool for mediation test (Preacher & Leonardelli, 2001). Sobel test is a significance test for the indirect effect of the independent variable on the dependent variable (Baron & Kenny, 1986). Furthermore, Hayes PROCESS model 4 was performed to establish whether the mediation effect truly was significant, using 1000 bootstraps.

Moderation effects

A moderator can be defined as a third variable that affects the direction or strength of association between an independent variable and a dependent variable. The term moderation refers to how a hypothesized causal relation between two variables is influenced by the moderator variable. In analysis of variance, a moderator effect is understood as an interaction between an independent variable and a factor that affects the specific operation of said independent variable. In general, a moderator effect is present in the case of a significant interaction between the independent variable and the moderator variable (Baron & Kenny, 1986).

According to Baron & Kenny (1986), a product variable approach should be applied if the moderator and independent variable are continuous, and if it is presumed that the effect of the independent variable on the dependent, influenced by the moderator, varies linearly. The product variable approach examines the moderation effect by creating a product of the moderator and the independent and adding it to the regression model (Cleary & Kessler, 1982). Two regression analyses were performed to check the two proposed interactions

effects of the hypothesized model (ref. Figure 1). The first proposed interaction involves pain catastrophizing moderating the association between pain intensity and pain-related disability. The second proposed interaction effect involves pain catastrophizing moderating the association between pain intensity and self-efficacy. First, the interaction variable (NRS and PCS) was computed. Following this, in step 1 for the first regression analysis, ODI was entered as a dependent variable and PCS and NRS as independent variables. In step 2, the interaction variable was entered. The second regression analysis consisted of GSE as a dependent variable and 0-10 NRS and PCS as an independent variable in step 1. In step 2, the interaction variable was entered as an independent variable.

In addition, the same analyses were performed using Hayes PROCESS model 1 using 1000 bootstraps.

Moderated mediation

The final statistical analysis performed was a moderated mediation analysis to examine our research question regarding how pain catastrophizing and self-efficacy might contribute in the association between pain intensity and pain-related disability. In short, moderated mediation analysis combines mediation and moderation to explain how a potential moderator variable may influence a mediated association between independent variables and a dependent variable (Hayes, 2018). In this study, the proposed hypothetical model (ref. Figure 1) was examined to investigate whether the association between pain intensity and pain-related disability was mediated by self-efficacy, as well as the potential moderator effects of pain catastrophizing on this possible mediation and the direct association between pain intensity and pain-related disability. PROCESS model 8 was selected to test the hypothesized model. Simple slopes analysis was also applied to follow up potential interaction effects at -1SD, mean and +1SD and 1000 bootstraps was used.

Ethics

The current study is categorized as a quality improvement study and does not fall under the mandate of Regional Committees for Medical and Health Research Ethics (REC). Therefore, it does not require a separate ethical evaluation and approval.

The OPR's research database includes data from patients who voluntarily sign a written informed consent. All consenting patients receive an encrypted link on e-mail requesting follow-up data at 12 and 36 months after their first consultation at the pain clinic. Data from

non-consenting patients are used exclusively for clinical purposes. The data from the OPR are stored on a server only accessible to the leader of the registry, Lars-Petter Granan. The data is stored with an encrypted patient identifier in accordance with approval from the local Data Protection Officer, thus making it impossible to trace the data to specific informants. In accordance with the principle of minimizing harm to participants, the participants were given a choice of completing the full version or a short version of the questionnaires.

In addition to ethical concerns regarding data collection and storing of the data, research on chronic pain gives rise to a few other ethical concerns such as how the research and the results may be interpreted by the general public. In this particular study, the research is concerned with how a small selection of psychological traits or variables might affect pain-related disability. Examining if certain psychological traits are advantageous or disadvantageous for pain related disability might unintentionally communicate that chronic pain is partly or fully produced by the patient, or even exaggerated by the patient.

In the context of the biomedical model, research indicate that a widely held belief is that a disease is real or legitimate only if there is evidence of observable tissue damage (Eccleston & Crombez, 2007; Newton, Southall, Raphael, Ashford & LeMarchand, 2013). Consequently, patients suffering from nonmalignant pain often report stigmatization when psychological explanations for their pain condition are adopted (Newton et al., 2013). Due to the lack of observable tissue damage, there is a specific risk that patients, professionals, and the general public might believe that the research question suggests that chronic pain is purely psychological, and therefore that chronic pain patients themselves are to blame for their pain condition. Such misinterpretations must be considered exceptionally harmful for an already stigmatized and marginalized group of patients. The biomedical model has challenged our possibilities to precisely understand the mechanisms behind chronic pain. The biopsychosocial model (Engel, 1977) does, however, suggests that biological, social and psychological factors are of great importance in causing and maintaining chronic pain. In pain research, it is then important to highlight the holistic view presented by the biopsychosocial model as this communicates that medical and psychological factors are equally legitimate factors involved in the mechanisms behind chronic pain.

3. Results

Descriptive analyses were performed to examine sample characteristics and the distribution of scores for the scales included in this study. A total of 2.156 participants identified as female, whereas 1.489 identified as male. The remaining 94 patients did not report their gender; however, the frequencies undoubtedly confirm an overrepresentation of female participants. All participants were in the ages of 16-97 years, with an average duration of pain of 8.06 years. Of all the 3.572 participants who answered the question regarding occupational status, 34.2 % reported that they were currently working, studying, or in military service, whereas 61.4 % reported that they were currently unemployed. Sample characteristics and descriptive statistics for the ODI, NRS, GSE and PCS are presented in Table 2 and Table 3. Note that the recoded variable of GSE (reversed scores) was included instead of the original GSE variable.

Table 2

Sample characteristics for ODI, NRS, GSE and PCS

Characteristics	*N (%)* $N_{total} = 3.739$	*Mean (range)*	*SD*
Age	3.739 (100%)	49.73 (16-97)	15.62
Duration of pain (years)	3.426 (91.6%)	8.06 (0-73)	9.08
Sex	3.645 (97.5%)		
Female	2.156 (56.6 %)		
Male	1.489 (39.8 %)		
Education	3.637 (97.3%)		
Comprehensive school (1-10 years)	554 (14.8%)		
Secondary/vocational school (11-13 years)	1621 (43.4%)		
College (14-17 years)	1135 (30.4%)		
Higher university degree (>17 years)	327 (8.7%)		
Employment/student	3572 (95.5%)		
Employed/Student/Military Service	1277 (34.2%)		
Unemployed	2295 (61.4%)		
Social benefits	3099 (82.9%)		
Retirement pension	484 (12.9%)		
Sick leave	363 (9.7 %)		
Work assessment allowance (AAP)	1000 (26.7%)		
Disability pension	1048 (28.1%)		
Unemployment benefits	18 (0.5%)		
Social benefit	64 (1.7%)		
Others	122 (3.3%)		

ODI = Oswestry Disability Index; NRS = 0-10 NRS Usual Pain Intensity; GSE = General self-efficacy scale; PCS= Pain Catastrophizing Scale

Table 3

Descriptive statistics for ODI, NRS, GSE and PCS

Questionnaire	*Mean*	*SD*
ODI	42.75	17.54
NRS	7.19	1.79
GSE	21.35	5.53
PCS	22.75	13.10

ODI = Oswestry Disability Index; NRS = 0-10 NRS Usual Pain Intensity; GSE = General self-efficacy scale; PCS= Pain Catastrophizing Scale

Results: correlation analysis

The results from the Pearson correlation analyses are presented in Table 4. The results showed a significant correlation between pain intensity and pain-related disability, $r = .42$, $p < .001$. Significant correlations between the independent variables were found; self-efficacy and pain intensity, $r = .145$, $p < .001$, self-efficacy and pain catastrophizing, $r = .395$, $p < .001$ and pain intensity and pain catastrophizing, $r = .349$, $p <. 001$. Note that the recoded GSE scale (reversed) was included and not the original GSE variable in the correlation analysis, thus the positive correlational relationship signifies that lower self-efficacy is associated with higher pain catastrophizing and pain intensity.

Table 4

Pearson correlation coefficients, all variables

	ODI	GSE	NRS	PCS
ODI	1	.307**	.421**	.379**
GSE	.307**	1	.145**	.395**
PI	.421**	.145**	1	.349**
PCS	.379**	.395**	.349**	1

ODI = Oswestry Disability Index; NRS = 0-10 NRS Usual Pain Intensity; GSE = General self-efficacy scale; PCS= Pain Catastrophizing Scale

** p < .001

Results: mediation analysis

The results from Baron & Kenny's (1986) method was further corroborated with Hayes Process in SPSS as well as the Sobel test. The results showed a significant direct effect of Pain Intensity (NRS) on the Oswestry Disability Index (ODI), b = .35, 95% CI [0.24 – 0.45].

The relationship between pain intensity and pain-related disability is depicted in Figure 2. A total effect of b = 4.00, p < .001 was derived from the analysis.

Figure 2

Path diagram illustrating the total effect between the independent and dependent variable

Total effect: b = 4.00, p < .001

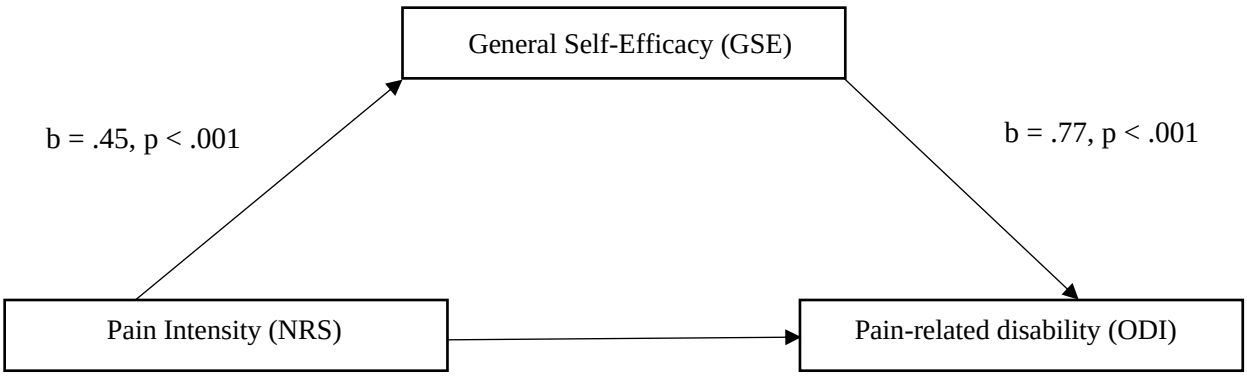
R^2 = .021

Figure 3 represents the proposed mediation analysis, wherein our results showed that pain intensity score is related to General Self Efficacy (GSE) score, b = .45, p < .001. Furthermore, the results from our analysis suggests that our proposed mediator, self-efficacy, is related to pain-related disability, b = .77, p < .001. A comparison of the total effect and the direct effect reveals that the association between pain intensity and pain-related disability is reduced when self-efficacy is included as a mediator, indicating that a significant partial mediation effect is present, albeit small-moderate. This is further corroborated by observed increase in the R^2 value when including self-efficacy as mediator between pain intensity and pain-related disability, R^2 = .021 without mediator variable versus R^2 = .24 with the mediator variable. In this specific case, the R^2-values reveal that reported pain intensity alone might explain 2.1% of the variance in pain-related disability, demonstrating that 97.9% of the variability in pain-related disability must be accounted for by other variables. Including self-efficacy in the model increased R^2 to 24%, signifying that the combined influence of pain intensity and self-efficacy better explains pain-related disability than pain intensity does in isolation, however with 76% of the variability still unaccounted for.

The Sobel test was conducted in order to verify that the results showed a consistent mediation effect. The Sobel test established a significant indirect effect of z = 2.18, p = .03. Running the analysis with Hayes PROCESS model 4 gave identical results.

Figure 3

Path diagram of the mediation model, including the results from the mediation analysis



Direct effect: b = 3.65, p < .001

Indirect effect: b = .35, 95% CI [0.24 – 0.45]

R^2 = .24

Results: moderation analyses

Two simple independent moderation analyses were performed using hierarchical regression with each dependent variable. Neither analysis resulted in a significant interaction effect, hence there was no real indication to assume a moderated mediation effect either. Running the moderation analyses with PROCESS model 1 generated identical results. Note that the insignificant interaction term (pain intensity x pain-related disability) in both moderation analyses showed high multicollinearity (highest VIF-value: 24.804 and lowest tolerance-value: 0.040). The results from the hierarchical regression analyses are presented in Table 5 and Table 6.

Table 5

Results from moderation analysis with PCS moderating the relationship between NRS and ODI

Variables	b	SE B	t	*P*	R^2	R^2 change
Step 1						
Constant	10.931	1.086	10.066	$p < .001$	.226	.226
NRS	3.325	.154	21.562	$p < .001$		
PCS	.303	.021	14.208	$p < .001$		
Step 2						
Constant	11.183	1.959	5.708	$p < .001$	.226	.000
NRS	3.290	.273	12.038	$p < .001$		
PCS	.290	.086	3.376	$p = .001$		
NRS x PCS	.002	.011	.155	$p = .887$		

Dependent variable = Oswestry Disability Index (ODI)

NRS = 0-10 NRS Usual pain intensity; PCS = Pain Catastrophizing Scale; NRS x PCS = 0-10 NRS Usual Pain intensity and Pain Catastrophizing Scale

Table 6

Results from moderation analysis with PCS moderating the relationship between NRS and GSE

Variables	B	SE B	t	P	R^2	R^2 change
Step 1						
Constant	17.217	.382	45.075	$p < .001$	.156	.156
NRS	.025	.055	.464	$p = .643$		
PCS	.171	.008	22.023	$p < .001$		
Step 2						
Constant	16.848	.700	24.068	$p < .001$	.156	.000
NRS	.077	.099	.781	$p = .435$		
PCS	.189	.031	6.162	$p < .001$		
NRS x PCS	-.002	.004	-.629	$p = .529$		

Dependent variable: General Self-efficacy (GSE)

NRS = 0-10 Usual pain intensity; PCS = Pain Catastrophizing Scale; NRS x PCS = 0-10 NRS Usual Pain intensity and Pain Catastrophizing Scale

Results: moderated mediation analysis

A moderated mediation analysis was performed with Hayes PROCESS using model 8, despite of the lack of indications of a possible moderated mediation effect. As predicted from the simple moderation analyses, no significant moderator effects were found. The interaction variable, NRS x PCS, was found to be insignificant, b = .0137, p = .2273. Additionally, R^2 change was .0004, further demonstrating an almost nonexistent accountability of the moderator on the dependent variable.

Based on the results it is inferred a partial mediation effect in the relationship, with GSE partly mediating the relationship between NRS and ODI. Furthermore, the results showed no significant moderator effects, neither for the simple regression analyses nor the moderated mediation analysis.

4. Discussion

The study in question aimed to explore possible mediation and moderation effects of selected psychological factors using Baron & Kenny's (1986) method for mediation, a product variable approach for moderation (Cleary & Kessler, 1982), as well as a moderated mediation based on Hayes PROCESS (Hayes, 2018). The factors hypothesized as possible mediator and moderators were drawn from the current body of research, with emphasis on the fear-avoidance model and the transactional model of stress. These models point towards a fear and threat related contribution to pain-related disability, such as pain catastrophizing. In addition, research investigating links between perceived self-efficacy and pain coping indicate that self-efficacy might facilitate the processes of pain relief by enhancing the tendency to select advantageous strategies for pain alleviation, as well as reducing the tendency to pain catastrophize and emphasize distressing anticipations that may exacerbate pain (Bandura et al., 1987; Lazarus & Folkman, 1984; Lethem et al., 1983; Perry & Francis, 2013). Previous research has revealed low to high correlations between pain intensity, pain catastrophizing, self-efficacy and pain-related disability (Jackson et al., 2014b; Karasawa et al., 2019; Perry & Francis, 2013; Scholich, Hallner, Wittenberg, Hasenbring & Rusu, 2012). Our correlational analysis revealed similar results, ranging from low to moderate correlations.

Based on these models and research findings, we hypothesized a model in which pain catastrophizing was suggested to be a moderator, and self-efficacy was suggested to be a mediator. Four separate sub-hypotheses derived from our hypothesized model: (1) there is a positive correlation between pain intensity and pain-related disability, (2) self-efficacy mediates the association between pain intensity and pain-related disability, (3) pain catastrophizing moderates the association between pain intensity and pain-related disability, and (4) pain catastrophizing moderates the association between pain intensity and self-efficacy. Lastly, to test our main hypothesis we examined the possibility of pain catastrophizing moderating the possible mediated relationship wherein self-efficacy was thought to mediate the association between pain intensity and pain-related disability.

The present study revealed a moderate positive correlation between pain intensity and pain-related disability, supporting the first hypothesis. The results from the correlational analysis are consistent with several earlier studies demonstrating significant positive correlations between some measure of pain intensity and pain-related disabilities for patients with

different pain locations although results have ranged from low to high correlations (Grönblad et al., 1996; Kovacs et al., 2004; Scholich et al., 2012).

Our results supported the second hypothesis, revealing that self-efficacy partly mediates the association between pain intensity and pain-related disability. This is in accordance with previous research reporting a partial mediation between pain intensity and pain-related disability with self-efficacy operating as a mediator (Arnstein, 2000; Arnstein et al., 1999; Costa et al. 2011). Woby, Urnston & Watson (2007) performed a study in which they found that self-efficacy mediated the relationship between pain-related fear and disability. In line with the results from this study, they also suggested that self-efficacy might partly mediate an association between pain intensity and disability. Similarly, Shelby et al. (2008) found that self-efficacy for physical function fully mediates the relationship between pain catastrophizing and physical disability. Baron & Kenny (1986) argues that partial mediation could indicate that several mediators are involved in predicting the outcome variable. Several studies also support that self-efficacy partakes in the regulation of pain coping and stress management. Drawing on this, the results may in a wider context indicate that individuals with stronger self-efficacy have a tendency to engage more in healthy behaviors, maintain healthy behavior, and to recover from difficulties (Luszczynska, Scholz & Schwarzer, 2005). Taken together, there is reason to assume that patients' beliefs concerning their pain coping abilities is involved in predicting pain intensity, pain behavior, and pain-related disability, however plausibly in a more complex context than the one hypothesized in this study.

As for the third and fourth hypothesis, the results proved to be in disfavor of pain catastrophizing contributing as a moderator in any of the two proposed interactions. Specifically, our results gave no significant indication of pain catastrophizing moderating neither the association between pain intensity and pain-related disability nor the association between pain intensity and self-efficacy. Correspondingly, we also found no support for our main hypothesis of a moderated mediation when checking for the hypothesized model as an integrated model. We evaluate some probable reasons as to why the hypotheses were disproven.

The null results: possible explanations

To our knowledge, no previous study has examined the specific relationships as proposed in our hypothetical model. Consistent with earlier research the current study revealed a partial mediation effect of self-efficacy in the relationship between pain intensity and pain-related

disability. On the other hand, our results revealed nonsignificant moderator effect and nonsignificant moderated mediation associations between pain-related intensity, pain intensity, self-efficacy and pain catastrophizing.

In the context of the current study, the nonsignificant moderator effects offer support to reject the possibility that there exists a combined influence of pain catastrophizing and pain intensity on pain-related disability, or a combined influence of pain catastrophizing and pain intensity on self-efficacy. The specific interpretation of the null result is that neither self-efficacy nor pain-related disability are attributable to a collective influence of pain intensity and pain catastrophizing.

If the results had supported the hypothesis of pain catastrophizing moderating the association between pain intensity and pain-related disability, it would have demonstrated that the tendency to pain catastrophize affects the strength in the relationship between pain intensity and pain-related disability. Specifically, a greater tendency to pain catastrophize would result in a stronger association between pain intensity and pain-related disability, whereas a low levels of pain catastrophizing would result in a weaker association between pain intensity and pain-related disability. Hence, revealing pain catastrophizing as a moderator would have demonstrated that the nature of the relationship between pain intensity and pain-related disability changes depending on the specific degree of pain catastrophizing. In the present study, a null result then signifies that the degree of pain catastrophizing does not significantly change the association between pain intensity and pain-related disability. The same would apply for the relationship between pain intensity and self-efficacy. Null results relating to the hypothesized moderator effects could simply imply that there is a less complex association between pain intensity and pain-related disability than the one postulated in our hypothesized model, i.e. an association in which no interaction effect exists. Alternatively, there might exist an interaction effect involving some other moderator variable not included in the current study.

As earlier discussed, the chronic pain literature provides solid evidence for the specific associations in our hypothesized model. Disproval of our hypothesis was therefore somewhat unexpected. Another possible explanation for the null results might then be that the importance or significance of pain catastrophizing has been overestimated in some contexts of chronic pain. This may in large relate to the fact that academia in general is affected by the large and increasing publication bias – a tendency to retain negative or undesirable results

from publication due to the perceived benefits of predominantly submitting positive or desirable results (Joober, Schmitz, Annable & Boksa, 2012). Publication bias poses a real threat to scientific advancements as exclusively presenting favorable results prevents an unbiased update of the credibility of the examined relationships. A biased body of literature diminishes the credibility of hypothesis testing due to new hypotheses being based on a false premise or a distorted understanding of the scientific framework (Joober et al., 2012). This highlights the importance of further examining the specific relationship between the factors included in our hypothesized model, as well as the presence of other relevant variables that may explain the remaining variance of pain-related disability.

Alternative approaches for future research

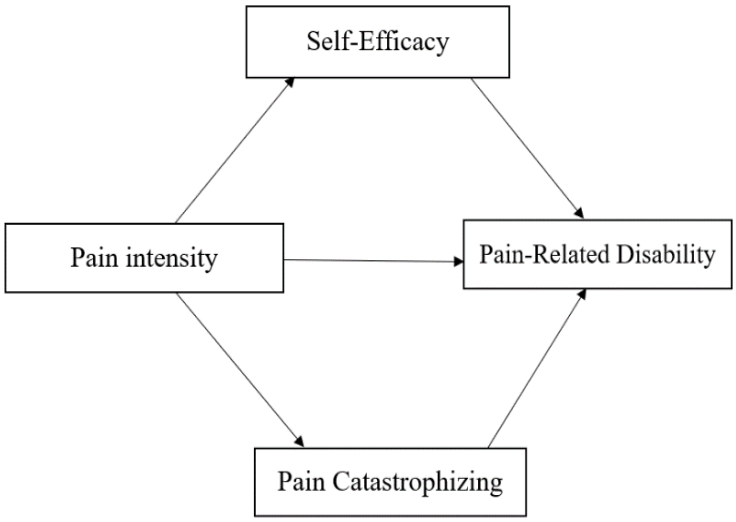
Notably few have examined pain catastrophizing as a potential moderator between pain intensity and pain-related disability, and pain intensity and self-efficacy. One similar study with an alternative understanding of the specific variable associations did however reveal a significant moderator effect of pain catastrophizing in the physical activity - pain intensity association (Lazaridou et al. 2019). Their study demonstrates pain catastrophizing as a potential moderator in some sort of association between physical capability and pain intensity. Despite this, their study is not directly comparable to the current study. Firstly, the study measured levels of physical activity in a sample suffering from a physical condition. Secondly, they chose pain intensity as an outcome variable instead and physical activity as the predictor, whereas our study examined the opposite. Thirdly, pain-related disability includes a larger variety of activities compared to physical activity, e.g. daily grooming, sitting, lifting, travelling etc. These differences might explain why the current study did not find any significant moderator effects; our results may be attributable to the chosen variable arrangement. Nevertheless, like Lazaridou et al. (2019), we argue that our results might encourage future research to think outside of the traditional assumptions regarding the psychological mechanisms involved in chronic pain.

As briefly mentioned, one possible explanation for the non-existent interaction effect in our hypothesized model is the specific arrangement of the selected variables. Previous studies have been inconclusive regarding the specific role of pain catastrophizing in pain-related disability, with some studies suggesting that pain catastrophizing plays a part as a mediator, and others suggesting a role as a predictor. With this in mind, we are left with two plausible explanations as to why the current study did not report a significant impact of pain catastrophizing. One possibility might be that pain catastrophizing actually operates as a

mediator, as it did in Smeets, Vlaeyen, Kester & Knottnerus (2006). The group performed a randomized controlled trial involving patients suffering from low chronic back pain, demonstrating that pain catastrophizing operated as a mediator between pain intensity and disability (Smeets et al., 2006). From research on the potential mediating effect of pain catastrophizing one might draw the conclusion that an alternative to our hypothesized model could be a model in which pain catastrophizing is included as an additional mediator in the pain intensity-disability relationship rather than a moderator. In this alternative hypothetical model, the relationship between pain intensity and pain-related disability consist of two parallel mediated pathways; one pathway mediated by self-efficacy, and the other pathway mediated by pain catastrophizing (Hayes, 2018). An alternative model wherein both self-efficacy and pain catastrophizing mediate the association between pain intensity and pain-related disability is depicted in Figure 4.

Figure 4

A parallel multiple mediator model hypothesizing self-efficacy and pain catastrophizing as potential mediators



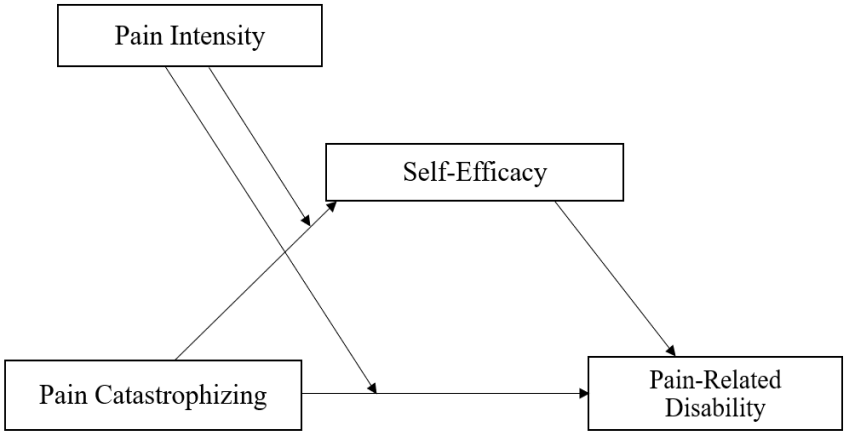
A second possibility might be that pain catastrophizing better explains pain-related disability as a predictor rather than a moderator or a mediator. Numerous studies support the hypothesis of pain catastrophizing as a significant predictor of pain-related disability (Arnow et al. 2011; Shelby et al. 2008; Sullivan, Stanish, Waite, Sullivan & Tripp, 1998; Sullivan et al., 2001). Based on their findings, Arnow et al. (2011) concluded that pain catastrophizing was a

significant predictor of pain-related disability. Shelby et al. (2008) performed a path analysis revealing that higher levels of pain catastrophizing negatively influenced self-efficacy for pain control, physical function and psychological symptoms. As mentioned, their study also demonstrated that domain-specific self-efficacy mediated the relationship between pain catastrophizing and disability. Based on this, the researchers suggested that the specific impact of pain catastrophizing on pain and disability is contingent on the patients' pain related self-efficacy and self-efficacy for physical function. Their study then supports an idea that catastrophizing is more likely to be a predictor of pain and disability rather than a mediator or moderator of pain and disability. This is in accordance with the perspective offered by the fear-avoidance model, where hypervigilance to pain or a catastrophic misinterpretation of pain contributes to increased pain-related disability (Gheldof et al., 2010).

The same idea has been studied by Suso-Ribera, Palacios, Botella & Canudas (2017) by examining pain intensity as a moderator between pain catastrophizing and psychical health outcomes such as performance in daily activities. The group concluded that the relationship between daily functioning and pain catastrophizing significantly varied as a function of pain intensity. It should however be noted that they applied the Coping Strategies Questionnaire (CSQ-C) as their measure of pain catastrophizing, a subscale that has been questioned regarding its validity (Sullivan & D'Eon, 1990). In addition to the somewhat questionable validity of their selected scale, the reader should also note that the present study did not include the subscale from CSQ-C but rather the Pain Catastrophizing Scale. Furthermore, Woby et al. (2004) reported that a higher level of pain catastrophizing was not significantly associated with greater pain intensity. Although they also selected the Coping Strategies Questionnaire (CSQ-C) as their measure of pain catastrophizing, their results still highlight the contradictions in the chronic pain literature regarding the association between pain intensity and pain-related disability. On that note, the current body of research also suggests that pain catastrophizing could be acting as a predictor rather than a mediator or moderator. A suggestion for a model with pain catastrophizing as an independent variable is illustrated in Figure 5. We suggest that future research examine this hypothesized association.

Figure 5

A hypothesized moderated mediation model including pain catastrophizing as an independent variable



Quartana et al. (2009) proposes a multi-process framework for studying pain catastrophizing in future research. They propose several relevant moderators for the association between pain catastrophizing and pain-related outcomes. They especially highlight genetics, family history, pain duration, psychiatric disorders and other types of negative cognitions associated with catastrophic thinking as probable moderators of pain catastrophizing on pain-related outcomes. Researchers propose that this view is likely to be more helpful in improving formulation of therapeutic interventions rather than focusing on the debate of classifying pain catastrophizing as a negative appraisal or as a coping strategy (Quartana et al., 2009). In their meta-analysis, Martinez-Calderon et al. (2019) argued that consistent reports of small associations between pain catastrophizing, pain intensity and disability gives an indication of a presence of several moderator effects in this specific relationship. In other words, how pain catastrophizing relates to the pain intensity-disability association is complex and as of date still highly uncertain. These findings clearly highlight the importance of examining additional variables and how they might relate to pain-related outcomes. The OPR includes a wide variety of relevant variables that can be included in future research aimed at examining the potential mediators and moderators associated with pain-related disability. For example, a future study based on the OPR dataset might include the Hopkins Symptom Checklist-25 as a potential moderator of pain catastrophizing in the pain intensity-pain related disability association, in accordance with suggestions from Quartana et al. (2009) regarding the potential role of psychiatric disorders moderating pain catastrophizing.

The relatedness of additional constructs of pain catastrophizing

Previous research indicates that pain catastrophizing is somewhat related to other constructs, such as depression, anxiety, anxiety sensitivity and fear of pain (Quartana et al., 2009). A study found strong associations between pain catastrophizing and depression and anxiety, questioning the distinctiveness of pain catastrophizing (Hirsh, George, Riley & Robinson, 2007). Similarly, a study using the Pain Catastrophizing Scale reported high associations between pain catastrophizing and fear of pain, state and trait anxiety (Sullivan et al., 2004), in which they also concluded that pain catastrophizing as a construct might not be as unique as originally assumed. Rather, it might be the case the pain catastrophizing actually represents a broader construct that overlaps with psychological variables. A high but not perfect correlation between pain catastrophizing and other constructs such as anxiety indicates that the variables highly represent the same construct, although not identical. The current study did not control for possibly overlapping constructs, and as such, it cannot be excluded that there could be a very small, but significant moderator effect relating to the unique elements of the pain catastrophizing construct. On the other hand, the opposite might also be possible; the components that are unique for pain catastrophizing might undermine the effects of overlapping constructs and therefore account for the insignificant moderator effect.

Study designs applied in chronic pain research

In line with methodological limitations reported in the pain literature, researchers argue that prospective study designs should be applied to a greater extent to confirm the specific directional relationships between pain catastrophizing, pain intensity and pain-related disability (Martinez-Calderon et al., 2019; Wideman et al., 2013). For instance, Martinez-Calderon et al. (2019) mentioned that their review mostly included cross-sectional studies. One of the defining characteristics of a cross-sectional study is that the variables of interest are examined at one point in time (Bordens & Abbott, 2014), critically limiting the conclusions regarding the specific directional relationship between the variables. Martinez-Calderon et al. (2019) suggests that prospective cohort studies, which involves following the participants over time examining how the variables to a certain outcome are connected, should be applied to a greater extent. This is supported by Wideman et al. (2013), arguing that the directional relationship between e.g. pain catastrophizing, pain-related fear and avoidance behaviors in the fear-avoidance model are highly recommended to be confirmed through prospective study designs. As the majority of previous research, the current study applied a similar study design. The inconclusiveness of the directional relationship between the

mentioned variables highlights the need for alternative study designs (e.g. prospective study designs) to further examine the specific ways the selected variables relate to each other.

Reflections upon the partial mediation effect

As mentioned, a partial mediation effect could indicate the presence of several mediators (Baron & Kenny, 1986). It is possible that the remaining variance in the association between pain intensity, pain-related disability and self-efficacy are additionally accounted for by other factors. For instance, Perry & Francis (2013) reported that self-efficacy is associated with lower levels of depression. A longitudinal study of low back pain (LBP) examined the mediating role of components of psychological distress such as depression, anxiety and stress in the relationship between pain intensity and later disability. This study reported a significant partial mediation effect of depression and stress, but not anxiety, in this particular relationship (Hall et al., 2011).

Furthermore, a proposed model by Pincus, Vogel, Burton, Santos & Field (2006) suggested that it is plausible for patients suffering from back pain to have coexisting clinical depression, unrelated to the pain condition, characterized by lack of energy, passivity and withdrawal. In a mixed sample of chronic pain patients in primary care, Arnow et al. (2011) reported a significant association between pain-related disability and depression. Hall et al. (2011) suggest that depression characteristics such as loss of self-esteem and low positive affectivity could impact a patient's involvement in daily activities resulting in greater disability. Additionally, these symptoms might contribute to pain chronification, while anxiety symptoms might have a greater impact in the acute stages of pain characterized by fear and autonomic arousal. Based on this, it is possible that depression symptoms such as low self-esteem impacts a patient's self-efficacy and consequently the pain intensity-disability association. However, the proposed link between self-esteem and self-efficacy and how it relates in the pain intensity-disability association should be examined in future research.

Methodological strengths and limitations

The main strength of the current study includes the large sample size of a heterogeneous group of chronic pain patients. To our knowledge, few previous studies have reported a sample size of outpatients with chronic pain conditions as substantial as the current study. The high response rate from patients with different pain locations strengthens the external validity of the current study, which in turn provides a better foundation to generalize the results to several populations of chronic pain patients. In terms of internal validity, the questionnaires

used in this study have been extensively tested and have evidently shown to be robust in terms of validity and reliability (e.g. Ferreia-Valente et al., 2011; Granan et al., 2019; Leganger et al., 2000; Osman et al., 1997; Vianin, 2008). Furthermore, the current study applied both Baron & Kenny's (1986) and Hayes's (2018) statistical approaches to mediation and moderation as well as conditional process analysis to test for a moderated mediation. The robustness of the results is considered strengthened due to the several different statistical approaches revealing identical results.

The cross-sectional nature and application of regression analyses severely limits conclusions regarding causality and the directional relationships between the variables. As previous mentioned, a longitudinal study design could offer a more robust method of examining this concern. Although most studies have examined pain intensity as a pain-related outcome variable, former cross-sectional studies (Arnstein, 2000; Costa et al., 2011) and the present study supports the idea of pain intensity as a predictor of pain-related disability. To date, there is no firm evidence regarding the specific direction or causality in the reported correlation between pain intensity and pain-related disability. Future longitudinal studies are critical for disentangling the directional relationship between pain intensity and pain-related disability. One possibility is to test the proposed parallel multiple mediator model, as depicted in Figure 4, in a longitudinal study.

It is worth noting that high multicollinearity was present in both moderator analyses when the interaction term (pain intensity x pain catastrophizing) was included. Nevertheless, Hayes (2018) argues that high multicollinearity is not of a great concern due to the p-value of the interaction term not being affected by the multicollinearity (Hayes, 2018).

One should note that the sample was predominantly female, however our sample included 1.489 male participants therefore we do not consider the gender skewness as an issue in terms of generalization. Moreover, our sample is based entirely on Norwegian patients residing in eastern and southern parts of Norway. As such, the results may be a subject of cultural and geographical variances both on a national and international basis.

Clinical implications

Despite the cross-sectional nature of the current study and other methodological limitations, we nevertheless argue that our results might contribute to a better understanding of the complex relationship between pain intensity, pain-related disability, self-efficacy and pain

catastrophizing in chronic pain patients. Additionally, we argue that our results might contribute with possible clinical implications and suggestions for areas for research.

Our results indicated a significant indirect effect of self-efficacy in the pain intensity-pain-related disability association. This might implicate the importance of assessing low self-efficacy in chronic pain patients and targeting it in psychological interventions. Previous research based on the fear-avoidance model has suggested the usefulness of cognitive-behavioral components in improving self-efficacy such as graded exposure to activities and tasks patients avoided due to fear of re-injury. Evidence indicates that with graded exposure, self-efficacy improved through experience of personal mastery of the activity and subsequently reduced fear of movement (Woods & Asmundson, 2008).

Cognitive-behavioral therapy (CBT) is widely acknowledged as an acceptable therapeutic intervention for chronic pain, including, but not limited to, targeting self-efficacy (Nash, Ponto, Townsend, Nelson & Bretz, 2013; Thorn, 2017). Even though treatment mechanisms are still not fully known, research generally establishes that changing patients' maladaptive cognitions are associated with lower pain intensity and distress (Thorn, 2017). In the context of self-efficacy, a study assessing the effect of CBT in a group of chronic pain patients in a pain rehabilitation center reported that psychoeducation on CBT, identification and restructuring of maladaptive beliefs and increasing positive responses to negative automatic thoughts (positive coping self-statements) increased patients rating of self-efficacy and decreased ratings of depression (Nash et al., 2013; Thorn, 2017).

Furthermore, an evidence-based psychological intervention for enhancing self-efficacy is the relaxation response (Darnall, 2019). This component is included in most of the psychological treatments of chronic pain, as well as CBT. Pain causes activation in the sympathetic nervous system, and a downregulation of this response can be achieved through deep breathing, and subsequently helps the patient to control the body's natural responses to pain, enhancing self-efficacy and thereby pain-relief (Metikaridis, Hadjipavlou, Artemiadis, Chrousos, & Darviri, 2016; Darnall, 2019). Additionally, acceptance and commitment therapy (ACT) has been shown to increase self-efficacy in chronic pain patients. Rather than focusing on changing maladaptive thoughts such as in CBT, ACT focuses on acceptance, mindfulness and commitment of patient's values and goals in the context of chronic pain (Darnall, 2019). A study found that for patients suffering from fibromyalgia reported greater self-efficacy, lower pain-related disability and decreased levels of depression after ACT intervention (Wicksell et

al., 2013). To summarize, our results are in favor of targeting and enhancing self-efficacy in psychological interventions where the overall aim is to improve and regain function.

Although the current study does not support the hypothesis of pain catastrophizing changing the strength of the relationship between pain intensity and pain-related disability, we did find evidence that pain catastrophizing correlates with pain-related disability. In addition, numerous studies indicate that pain catastrophizing predicts pain-related disability. This might justify a targeted focus on pain catastrophizing in psychological treatment, perhaps with a CBT approach (Darnall, 2019).

In general, clinicians should bear in mind that chronic pain patients represent a marginalized group and frequently experience stigmatization (Newton et al., 2013), such as the patients' experience of the health care system judging their pain condition as not being legitimate due to lack of observable evidence. This is important as chronic pain patients are reported as being less receptive to therapeutic interventions such as CBT when experiencing delegitimizing of their pain condition (Thorn, 2017).

5. Conclusions

Pain catastrophizing and self-efficacy have been pointed out as important psychosocial determinants for pain, and critical targets in the treatment and rehabilitation of chronic pain patients. The purpose of the current study was to investigate the specific roles and relationships of pain catastrophizing and self-efficacy on the association between subjectively evaluated pain intensity and the extent of pain-related disability. A main hypothesis along with four sub-hypotheses were tested; we assumed that the association between pain intensity and pain-related disability is mediated by self-efficacy and moderated by pain catastrophizing. Furthermore, pain catastrophizing was thought to moderate the link between pain intensity and self-efficacy. Firstly, the results confirmed a significant moderate correlation between pain intensity and pain-related disability, as several other studies have found. Secondly, in line with previous research, the results indicated that self-efficacy partly mediates the association between pain intensity and pain-related disability. Thirdly, the results gave no evidence for pain catastrophizing operating as a moderator neither in the association between pain-intensity and self-efficacy, nor between pain intensity and pain-related disability thus disconfirming two of our sub-hypotheses. Accordingly, the results were in disfavor of our main hypothesis of a moderated mediation effect. The null results relating to the hypothesized moderator effects could simply imply that there is a less complex association between pain intensity and pain-related disability than the one postulated in our hypothesized model. Alternatively, there might exist an interaction effect involving some other moderator variable not included in the current study.

Previous studies have been inconclusive regarding the specific role of pain catastrophizing on pain-related disability, with some studies suggesting that pain catastrophizing operates a mediator, and others suggesting a role as a predictor. The combined knowledge from previous studies and the results from this study then suggests that pain-related disability might be better explained by an alternative arrangement of pain intensity, pain catastrophizing and self-efficacy. We suggest two alternatives to altering our hypothesized model; a) pain catastrophizing might actually operate as a mediator, thus changing our hypothesized model from a model of a moderated mediation to a parallel multiple mediator model, or b) pain catastrophizing might actually not be a mediator nor a moderator in the examined association, but rather the main predictor of pain-related disability. Research has yet to settle the specific directions in processes and mechanisms behind pain-related disability in chronic pain patients.

Longitudinal studies are recommended in order to achieve a more accurate understanding of how different types of variables are involved in determining pain-related disability in chronic pain patients.

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