

Emotional and cognitive influences on pain experience

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Emotional and Cognitive Influences on Pain Experience

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Abstract

Multiple emotional and cognitive factors impact on the experience of pain. This chapter will review some of the most important emotional and cognitive determinants of the pain experience as found in experimental and clinical studies with human participants. Emotional factors that may increase pain perception are anxiety, depression and anger. Positive emotions usually decrease perceived pain. The cognitive factors attention, expectancy and appraisal can either increase or decrease pain experiences depending on their specific focus and content. Many brain regions are involved in nociceptive processing and bringing pain into awareness. There are profound interconnections between areas processing sensory, emotional and cognitive information. Descending pathways from cortical areas to the midbrain and spinal levels can facilitate or inhibit spinal nociceptive information and thereby afferent nociceptive input to the brain. The underlying mechanisms of the various emotional and cognitive modulatory influences may partly overlap, but also have some unique aspects. What becomes clear is that pain is not merely a reflection of the nociceptive input, but should be considered as a complex experience shaped by psychological factors that may be unique for each individual.

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Pain is not solely determined by the degree of nociceptive input, but also depends on psychological factors such as emotional state and cognitive appraisal. The International Association for the Study of Pain (IASP) defines pain as ‘an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage’. According to this definition, pain is unquestionably a bodily sensation, but it is also always unpleasant and therefore an emotional experience [1]. Brain imaging research has demonstrated that sensory, affective and cognitive regions interact to determine the final pain experience. Several decades of research

have identified some of the most important emotional and cognitive determinants. This chapter will discuss successively: fear and anxiety, depression, anger and positive affect as the emotional components of pain, and attention, expectancy and appraisal as the cognitive components. Even though for the sake of this overview these aspects are treated separately, as we will see later, some of these components are inherently linked to each other. It should also be kept in mind that although the focus of this chapter is on emotions and cognitions as determinants of the pain experience, there is a reciprocal relationship. Moreover, as implied in the definition, emotions are also an integral part of the pain experience itself.

Emotional Factors

Before proceeding to reviewing emotional modulation of pain, a clarification of terms is in order. *Emotion* is usually conceptualized as a reaction to a particular elicitor and has a clearly defined focus. *Mood* is a more diffuse state without a clear focus. The term *affect* is used to refer to emotions and mood collectively. It should also be noted that emotional factors can refer to short-lasting states (e.g. after anger induction) or to longer-lasting states (e.g. clinical depression). The overview presented below will incorporate studies that have focused on both short- and long-lasting emotional states and moods.

Anxiety and Pain-Related Fear

Research in healthy volunteers using experimental pain stimuli has demonstrated that anxiety, specifically pain-related anxiety, can increase pain sensitivity and exacerbate the pain experience [2–6]. Additionally, in clinical situations anticipatory anxiety has been associated with higher pain reports. For instance, anxiety concerning injections led to higher reported pain after dental injections [7]. Rhudy and Meager [8] distinguished between the effects of fear versus anxiety. Anxiety, defined as the anticipation of potential threats, was proposed to lead to augmentation of the pain experience. Fear, defined as the alarm reaction to a present threat, was proposed to suppress pain experiences and to mobilize the body to allow escape. Their experiment confirmed these predictions: when moderately intense shocks (supposed to elicit fear) were delivered prior to a painful test stimulus, pain was inhibited; however, when threat of intense shocks was induced without actually giving these shocks, sensitivity for the test stimulus was enhanced [8]. The anxiety state would be most characteristic of the emotions experienced in most experimental studies and in clinical situations. Fear and inhibition of pain, on the other hand, may typically be seen in extreme situations such as during combat or assault, where pain reactions might hamper escape and endanger survival. In much of the literature this distinction between anxiety and fear is, however, not made and the term ‘pain-related fear’ is used to denote both the reaction to current pain as well as anticipatory anxiety for upcoming pain.

Pain modulation by anxiety can be traced back to brain interactions of emotional and sensory areas. Incoming nociceptive signals are processed in many different brain areas such as the somatosensory cortex, anterior cingulate cortex (ACC), insula, amygdala, thalamus and hypothalamus [9, 10]. Projections from the insula and prefrontal cortex, via periaqueductal gray (PAG) to brainstem nuclei, and finally to the spinal cord are involved in descending modulation of pain. This descending modulation can be both inhibitory and excitatory, and involves among other endogenous opioids, noradrenaline and serotonin [9–11].

Anxiety can directly or indirectly influence many different areas involved in coding sensory and affective aspects of pain as well as areas involved in descending modulation. For instance, one study found that conditioned anxiety increased activation of the entorhinal cortex of the hippocampal formation, which subsequently affected activity in the midinsula intensity coding area [12]. Many studies also point to the pivotal role of prefrontal areas, the amygdala and the PAG in conveying the influence of anxiety on pain. Anxiety-prone individuals show impaired prefrontal functioning, which is generally assumed to inhibit pain [9]. In addition, anxious people show a different connectivity pattern between the anterior insular cortex and the PAG, suggestive of less efficient pain modulation [13, 14]. This is not an exhaustive overview of imaging studies that have shown how anxiety might impact on the neural pain network, as a variety of mechanisms have in fact been identified [14].

Anxiety has also been implicated to play a role in chronification of pain. One of the most influential psychological models of chronic pain is the fear-avoidance model [15, 16]. This model has mostly been applied to chronic musculoskeletal pain. According to this model, fear of pain and fear of reinjury may hamper recovery from an acute pain episode because this anticipatory anxiety motivates avoidance behavior and inactivity. Persistent inactivity may be maladaptive and can lead to a negative spiral of deteriorated physical condition, disability and distress, and subsequently to increased pain intensity. Correlational studies have indeed confirmed the predicted relationships between pain-related fear and pain intensity, disability and distress in patients with musculoskeletal pain [17, 18]. More pertinent for its proposed etiological role, prospective studies have shown that pain-related fear in patients seeking care for acute pain may be predictive of pain persistence and long-term disability and sick leave [19–21]. Pain-related fear is also related to the future inception of (back) pain in the general population [22, 23].

A suitable model to study the transition from acute to chronic pain is the postsurgical pain model. Surgery causes tissue injury and massive nociceptive input. Depending on the specific procedure, patients experience acute postsurgical pain for a few hours to several days or even weeks after the operation, which for the majority of patients will subside in time. However, a minority of patients can go on to develop chronic postsurgical pain (CPSP). Prevalence estimates for CPSP have ranged from 5 to 30% for the most common surgical procedures, but can be as high as 50% for

amputations and thoracotomy [24, 25]. Because elective surgery is a planned procedure, it allows for the assessment of psychological variables before the intervention takes place. Studies on predictors of CPSP have identified general anxiety as well as surgery-specific anxiety as important predictors of long-term surgical outcome in terms of pain and disability [26, 27].

In conclusion, anxiety seems to aggravate the subjective experience of a nociceptive stimulus, it can trigger maladaptive behaviors in patients with chronic pain and it may predispose people to develop a chronic pain condition. As the next sections will demonstrate, anxiety is certainly not the only emotion affecting the pain experience. However, it is probably the most researched factor in the past decades with generally robust findings.

Depression

A vast number of studies are available demonstrating that negative emotions increase pain perception. Most studies examining the effect of negative emotion induction on experimental pain have not differentiated between the specific emotions that were elicited (e.g. disgust, anger, depression), with a few exceptions. Zelman et al. [28] used the Velten mood induction procedure, where participants read depressive statements, and found lowered tolerance for cold pressor pain in comparison to a neutral mood condition. Berna et al. [29] used the Velten procedure accompanied by sad music to induce a depressed mood and applied tonic heat stimuli in the scanner. Depressed mood induction led to higher perceived pain unpleasantness and increased activity in various pain-related brain areas including prefrontal and anterior cingulate cortices and the hippocampus.

Other studies have examined pain sensitivity and pain processing in patients with major depressive disorders. These studies show an inconsistent relation between clinical depression and pain sensitivity, with hypersensitivity, hyposensitivity or no differences between depressed and nondepressed participants [9]. Brain imaging studies also found contradictory results. For instance Bär et al. [30] found depressive patients have less sensitivity for thermal pain stimuli with concomitant increased activation of the ventrolateral and dorsolateral prefrontal cortices, which have a pain-inhibiting function. On the other hand, Strigo et al. [31] observed decreased activation in pain modulatory areas including prefrontal areas and the PAG during heat pain stimulation.

Clinical observation and epidemiological studies have demonstrated a strong association between chronic pain and depression. As much as 50% of patients with chronic pain suffer from a comorbid depressive disorder [32, 33]. Concurrent depressive symptoms in patients with chronic pain are associated with increased disability and an augmented pain experience [34, 35]. Imaging studies in patients with chronic pain have shown that concomitant depression is especially associated with increased activity in brain regions involved in affective processing both in response to experimental pain and disease-relevant pain [36, 37].

A long-standing debate is whether chronic pain causes depression, or whether depression is a risk factor for developing chronic pain. Both may in fact be true, with one condition constituting a risk factor for the development of the other condition. In addition, the co-occurrence of chronic pain and depression may be due to shared underlying mechanisms, either cognitive behavioral (e.g. repetitive negative thinking, avoidance [38]) or biological (e.g. neuroinflammation [39], genetic [40]). Longitudinal studies provide evidence that having depression might increase the chance of a subsequent pain disorder, which is consistent with the risk factor or shared vulnerability view. The incidence of developing back pain in previously pain-free individuals was found to be 2–3 times higher in individuals who suffered from a previous or current depressive disorder [41, 42]. Also with regard to the development of CPSP, pre-operative depression has been identified as a risk factor, although less consistently so compared to anxiety [43, 44].

Anger

Less frequently researched than the above two negative emotions is the influence of anger on pain. Nevertheless, evidence is accumulating that anger can also influence pain perception. Several studies have examined the effects of anger induction on pain sensitivity in pain patients or healthy controls and generally found increased sensitivity for experimental pain stimulation [45, 46]. In addition, anger management style may influence pain sensitivity. Especially individuals who are more prone to express anger in a physical or verbal way (i.e. anger-out style) show greater sensitivity, and this was found for experimental pain, as well as for acute postsurgical pain [47, 48]. It has been speculated that deficient endogenous opioidergic modulation in the rostral limbic system might underlie both increased pain sensitivity and a reduced ability to adequately regulate anger [48]. However, anger management style and state anger can show a complex interaction; when people with a high level of anger cannot express their anger (e.g. in a lab situation it is usually not appropriate to express anger), this may indeed increase sensitivity; however, when anger is expressed, this may in fact normalize or even decrease pain sensitivity [48, 49].

Some indication for a possible causal relation between anger regulation and increased pain comes from daily diary studies with chronic low back pain patients [49, 50]. Behavioral anger expression and anger inhibition were associated with increased pain intensity, more pain interference and decreased functioning at subsequent assessment times. In addition to a direct effect on the pain experience, anger may also have a negative effect on treatment outcome and lead to interpersonal problems [51, 52].

Positive Emotions

Although most research has focused on the role of negative emotions, several studies have specifically examined the effect of positive emotions on pain. It should be noted that positive emotions are not just the opposite of negative emotions, they are

correlated but can also vary partly independently [53]. Experimental studies in healthy volunteers have demonstrated that the induction of positive emotions leads to reduced pain sensitivity (for an overview, see Finan and Garland [54]). Again, both supraspinal modulation (i.e. interactions between pain and emotional networks within the brain [14]) and descending pain modulation are involved, as shown by a decrease in a spinal nociceptive reflex after positive emotion induction [55, 56].

Positive emotions may also have a protective role in the transition from acute to chronic pain because they may protect against the development and spreading of pain-related fear. In an elegant experiment, fear for a specific hand movement was experimentally induced by pairing this movement with electrical shock. During the extinction phase, where shocks were no longer given, participants with high trait positive affect showed faster fear reduction than participants with low positive affect [57]. A subsequent study showed that the induction of positive emotions could prevent spreading of fear to new movements and to lower pain expectancy for these movements [58].

In chronic pain patients, positive affect and pain intensity have repeatedly been found to be correlated [59]. However, many of these studies were cross-sectional and did not allow conclusions on the direction of causality. A few studies nevertheless suggested that positive affect may be prospectively related to less intense pain. For instance, a diary study indicated that an increase in positive emotions was related to lower pain reports at subsequent time points in patients with rheumatoid arthritis [60]. Other studies have indicated that positive emotions may buffer the effect of pain on physical functioning and distress. Positive emotions were related to faster functional recovery after hip fractures [61] and to less distress in patients with rheumatoid arthritis at times when pain intensity was high [62]. Induction of positive emotions may reduce clinical pain and distress. A randomized controlled trial studying the effects of an online positive psychology intervention on daily pain demonstrated that people reporting mild-to-moderate pain before the intervention showed significant reductions in pain at the 6-month follow-up [63]. Preliminary data from a study using virtual reality to induce positive emotions suggest that this intervention might decrease pain and distress, and increase activity level in fibromyalgia patients [64].

In sum, positive emotions may have pain-reducing qualities. This effect may at least be partly dependent on their incompatibility with pain-related fear and probably other negative emotions.

Cognitive Factors

In addition to emotional factors, cognitive factors also influence the perception and experience of pain. Cognition is defined as the mental processes related to gaining knowledge and encompasses such functions as attention, memory and reasoning. The cognitive processes most studied in relation to pain are attention, expectancy and appraisal.

Attention

Attention and nociception are intrinsically related and have a bidirectional relationship with each other [65]. Pain can involuntarily capture attention, especially when it is intense, novel or threatening [66]. A shift in attention towards the source of a potential threat enables escape and prevention from further harm. On the other hand, directing attention towards pain and bringing it into focal awareness may increase its intensity. In reverse, directing attention away from pain will exclude it from further processing and lead to less pain [67].

Numerous studies have documented the effect of attention manipulations on perceived intensity of experimental pain, with the general finding that distraction by a competing task diminishes pain and focusing on pain increases it [68, 69]. Brain imaging studies have shown that distraction away from pain is associated with decreased activity in ascending pain pathways, including the thalamus, somatosensory areas, insular cortex and ACC, and increased activity in descending pain modulatory pathways involving the ACC and PAG [70–72]. Animal studies have shown the involvement of dopamine, serotonin and endocannabinoids in attentional modulation of pain [73, 74]. However, involvement of the descending system in attentional control has recently been questioned as some studies suggest that the underlying neural mechanisms of attentional modulation may be partly distinct from those underlying the effects of emotion on pain [72]. When emotion and attention were independently manipulated, emotional influences on pain perception did involve the PAG and ACC, suggestive of descending modulation, but attentional modulation affected the superior posterior parietal cortex and the anterior insula [75]. The latter is consistent with orienting attention toward sensory information and optimizing the cortical presentation of the attended stimulus [74]. Nevertheless, even though attentional and emotional modulations of pain may originate from different cerebral sources and may partly act through different mechanisms, there are also communalities between their actions, e.g. both can modulate activity in the anterior insula [76].

Attentional processes also influence the experience of clinical pain. Heightened attention towards painful signals – also referred to as hypervigilance – have been implied in the transition from acute to chronic pain [16]. Such hypervigilance may especially become apparent in individuals that are highly fearful of pain and for whom avoidance of pain is the main goal [77, 78]. In patients with chronic pain, high pain vigilance was indeed found to be associated with higher pain intensity [79, 80].

Another line of studies has examined the effectiveness of distraction as a coping strategy for managing chronic pain. Although distraction is one of the most frequently used and recommended strategies for managing pain, results regarding its effectiveness remain variable [67]. Several factors may moderate the efficacy of distraction, e.g. motivational relevance of and engagement in the distraction task, pain intensity and habitual hypervigilance [78, 81]. Nevertheless, under proper circumstances, distraction may be a powerful analgesic technique. For instance, the use of virtual reality distraction has proven successful in reducing experimental, procedural and chronic

pain [82, 83]. Virtual reality involves multisensory information facilitating full immersion in the distracting environment. Experiencing a high level of presence in the virtual environment, as well as fun in interacting with the environment, determine the efficacy of virtual reality distraction [84].

Expectancy

A plethora of studies have been performed on placebo, and to a lesser extent nocebo, effects on pain, effects that are highly dependent on expectancies of pain relief or pain increases, respectively [85]. Verbal suggestion that pain relief or increase may follow can be sufficient for inducing strong expectations, and subsequently influencing the actual experience of pain [86]. Colloca et al. [87] demonstrated that the verbal suggestion that the intensity of weak electrocutaneous shocks would increase not only led to the judgment of stimuli that were previously just above the pain threshold to be judged as much more painful, but also that tactile stimuli (i.e. stimuli previously judged as nonpainful) could be turned into painful stimuli. Neuroscientific research has indicated that the effects of expectancy on the subjective pain experience are paralleled by changes in afferent nociceptive brain areas (e.g. somatosensory areas and insula) and that this is at least partly mediated by descending pain modulatory circuits (e.g. ACC and PAG) [88–90].

Expectations may also interact with the analgesic efficacy of drugs and with other pain modulatory mechanisms. Induction of positive treatment expectancy was shown to lead to a substantial increase in the analgesic effect of a potent opioid, while induction of a negative treatment expectancy completely abolished the analgesic effect [91]. This effect on pain perception was accompanied by changes in activity in the descending pain modulatory system. Cormier et al. [92] found further evidence that expectancies might work through descending modulation and affect nociceptive processes at the spinal level. They used a conditioned pain modulation procedure, i.e. the phenomenon that a noxious stimulus (the conditioning stimulus) reduces the perceived intensity of another noxious stimulus applied on a different part of the body (the test stimulus). Conditioned pain modulation has been found to depend on descending pathways from the brainstem and to inhibit spinal nociceptive processing [93]. The effect of the conditioning stimulus on pain perception as well as the spinal nociceptive flexion reflex was moderated by expectations. Nociceptive inhibition was enhanced when participants expected the conditioning stimulus to induce analgesia; however, when participants expected more pain after the conditioning stimulus, the prototypical inhibitory effect reversed and increased pain reports and elevated reflexes were found [92].

Despite the fact that placebo and nocebo expectations may act through the same central and spinal mechanism, there may be different underlying biochemical factors [86]. Expectations of pain relief were found to act through activation of the endogenous mu-opioid system [94, 95], whereas expectations of pain increase may be mediated by cholecystokinin [96]. The underlying mechanism of expectation-induced analgesia may also be different from the mechanisms involved in the effects of distraction

on pain [97], with expectation relying more on (opioid-dependent) descending pain modulation than distraction [74].

The induction of positive expectancies can also be clinically used, for instance by verbally stressing expected pain relief in patient-healthcare provider interaction. Negative expectancies about the effects of an analgesic treatment – by the patient as well as by the healthcare provider – may on the other hand reduce the efficacy of treatment [98].

Finally, expectancies may influence the recovery from acute pain and injury. Patients' expectations for recovery after traffic-related whiplash injuries have proven to be an important prognostic factor for neck pain intensity and limitations 1 year later [99]. In postoperative pain studies, expected pain and expected recovery were shown to predict actual surgical outcomes. Expectations of high acute postoperative pain were predictive of pain scores during the first postoperative days [100, 101]. Negative expectations about continuing pain, disability and ability to work led to continued pain and disability after lumbar disc surgery for back pain [102, 103]. Also for other operations, such as total knee arthroplasty, negative expectancies were found to be associated with CPSP [104].

Appraisal

More complex cognitive processes, i.e. our interpretation of the meaning of pain, its causes and consequences, also color the experience of pain. Most research has been devoted to one specific (negative) cognitive pain appraisal, catastrophizing. Catastrophizing is defined as an exaggerated negative mental set regarding actual or anticipated pain and encompasses several aspects of negative thinking: exaggeration of the threat value of pain, rumination about pain and feeling helpless in the face of pain [105]. Catastrophizing about pain has close interconnections to some of the concepts discussed above, most notably anxiety, attention and expectancy. According to the fear-avoidance model of pain discussed earlier, catastrophizing is the precursor of pain-related fear and of increased attention towards pain (i.e. pain vigilance) [15]. It also has aspects of expectancy, i.e. expectation that the pain may not go away or becomes worse. Not surprisingly, many of the same associations between catastrophizing and pain have been reported as were found for the constructs discussed above. In healthy individuals, for example, pain catastrophizing is related to increased sensitivity to experimental pain [105, 106], and in chronic patients to higher reported pain intensity, more pain interference and more disability [107]. Also, the pain-modulating influences of catastrophizing seem to be associated with similar brain regions that are implied in attentional and emotional control of pain [108, 109], and with lower activation of descending pain-inhibitory controls [110, 111].

Pain catastrophizing has also been implicated as an important factor in the transition from acute to chronic pain. Catastrophizing predicts the persistence of pain after an acute back pain episode [112] and persistent pain after surgical intervention [27, 113, 114]. A catastrophic thinking style can interact with genetic factors in determining persistent pain. The combination of a specific gene diplotype and high levels of

pain catastrophizing were found to increase shoulder pain intensity after exercise-induced muscle injury and shoulder surgery more than either factor on its own [115, 116]. Many more studies in fact found associations between pain catastrophizing and pain outcomes in both healthy volunteers and pain patients; there are several reviews available that summarize this evidence [105, 106, 117].

Another higher-order cognition that has been found to influence pain perception is optimism. Optimism, as well as its reverse pessimism, is related to catastrophizing yet distinct in that it concerns a broader thinking style affecting multiple life domains. It is usually thought of as a trait characteristic, remaining relatively stable across the life span [118]. An optimistic thinking style has been related to less pain sensitivity and to less interference of pain on activities and mood [119]. In laboratory experiments, trait optimism, as well as (temporary) induced optimism, was found to lead to lower perceived pain, and this effect seemed to be mediated by lower levels of pain catastrophizing [120, 121]. Optimism was also indicated as a protective mechanism for the development of chronic postoperative pain after surgery [122].

Concluding Remarks

The above review makes clear that multiple emotional and cognitive factors can impact on the experience of pain. Although the various factors were presented and discussed separately for the sake of this overview, it should be noted that there are profound interconnections between these factors. For instance, both pain catastrophizing and pain-related fear may lead to attention to pain; a general optimistic attitude may result in less fear and less pain catastrophizing, etc. In addition, the underlying pathways may partly overlap. Brain areas processing affective and cognitive information are heavily interconnected and in turn both have connections to sensory areas. Descending pathways play a role in many of the effects presented above. Probably the primary distinction that can be made is between emotion modulation in general (i.e. independent of the specific emotion involved) and attentional modulation because there are indications that for these two modulatory influences the underlying mechanisms may differ. The more complex cognitive factors such as pain catastrophizing and optimism probably rely on multiple underlying emotional (e.g. fear) and more basic cognitive factors (e.g. attention). For treatment and management of (chronic) pain, all of these factors might, however, constitute targets for prevention and intervention, or may be used for determination of risk for a negative pain trajectory. Discussion of treatment strategies for decreasing negative emotions and increasing positive emotions, and/or changing pain cognitions, is beyond the focus of this chapter but can be found elsewhere [123–126]. The principal message is that proper understanding of the experience of pain always involves awareness of emotional and cognitive aspects involved. Pain is a complex experience shaped by multiple factors that are unique for each individual.

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