Don’t Worry, This Will Only Hurt A Bit:

The Role of Expectation and Attention in Pain Intensity

Abstract

To cause pain, it is not enough to deliver a dose of noxious stimulation. Pain requires the interaction of sensory processing, emotion, and cognition. In this paper, I focus on the role of cognition in the felt intensity of pain. I provide evidence for the cognitive modulation of pain. In particular, I show that attention and expectation can influence the experience of pain intensity. I also consider the mechanisms that underlie the cognitive effects on pain. I show that all the proposed mechanisms of pain modulation affirm the view that cognition impacts the sensory and discriminative aspects of pain. I conclude that pain perception is a cognitively penetrated phenomenon.

1. Introduction

 Pain is a complex phenomenon with distinct features; there are sensory and discriminative aspects of pain, affective and motivational elements of pain, and then there are cognitive facets of pain. Although each of these elements is realized by distinct brain pathways, pain arises from the activation and interaction of all those brain mechanisms. Emotions, such as anxiety, can impact the felt intensity of pain, as can cognitive processes, such as expectation, attention, and learning. The cognitive effects on pain, in particular, have been well-documented in the scientific literature, giving rise to a consensus that many cases of cognitive modulation result in actual changes in the perceptual processing of pain.[[1]](#footnote-1) Recently, however, the claim that cognition directly affects perceptual processing has been challenged in relation to visual perception. [[2]](#footnote-2)

In this paper, I examine whether some of the criticism leveled against cognitive penetration of vision refute the view that cognition modulates pain perception and I conclude that it does not. In section 2 of the paper, I begin by outlining the gate control theory of pain, which distinguishes between the three distinct but interacting aspects of pain. I also outline evidence for each of the distinct elements of the pain system postulated by gate control theory. In section 3, I provide an abridged review of the support for cognitive modulation of sensory and discriminative aspects of pain. Specifically, I describe how expectation and attention can modulate the sensory and discriminative aspects of pain, such as pain intensity. In section 4, I assess the evidence for the neocortical modulation of pain in relation to the recent challenge to cognitive penetration of perception. I show that cognition can inhibit the processing of the sensory and discriminative aspects of pain. I argue that these changes are not mere shifts in attention, but amount to modifications in sensory processing of noxious stimuli. Finally, I argue that cognitive alterations in felt intensity of pain are not changes in judgment about what is perceived, but are actual changes in what is perceived. I conclude that pain perception is a cognitively penetrated phenomenon.

2. The Pain System[[3]](#footnote-3)

There are three proposed scientific theories of pain: specificity theory, intensity theory, and gate control theory. Of the three, gate control theory provides the most comprehensive account of a variety of pain phenomena and is currently the dominant scientific account of pain. Before describing the gate control theory of pain, I will briefly discuss the two alternative theories of pain to show how gate control theory succeeds where the competing views failed. In subsequent sections of the paper, I will utilize gate control theory to support my argument for the cognitive penetration of pain.

Intensity theory of pain is based on the rejection of dedicated nociceptive receptors (Melzack and Wall 1965 973).[[4]](#footnote-4) Instead, intensity theory is the view that pain is the result of stimulus strength and stimulus summation.[[5]](#footnote-5) This view was based on studies performed by Bernhard Naunyn in 1859, showing that when non-noxious stimuli were successively applied to the skin, they caused pain in syphilitic patients (Moayedi and Davis 2013 8).[[6]](#footnote-6) This evidence was thought to indicate that pain is the result of stimulus summation that has reached a threshold of strength sufficient to cause pain. Intensity theory, however, was replaced by specificity theory of pain once dedicated nociceptive receptors and fibers were identified (Melzack and Wall 1965 971-974).

Specificity theory incorporates the evidence for nociceptive receptors (Dubner, Sessle, and Storey 1978).[[7]](#footnote-7) Based on specificity theory, nociceptors relay information received from the periphery of the nervous system to a specific “pain center” in the brain, which was thought to be in the thalamus (Melzack and Casey 1968).[[8]](#footnote-8) One of the failings of specificity theory is that there is not a one-to-one relationship between noxious stimulation and pain. For example, pathological pain states that occur in absence of a noxious stimulus, such as causalgia, peripheral neuralgia, and phantom limb pain, undermine the claim that pain requires noxious stimulation. The phenomenon of pain asymbolia (which occurs when the unpleasantness of pain is absent despite the presence of a noxious stimulus), on the other hand, challenges the view that all noxious stimulation results in pain. For example, individuals who have pain asymbolia can appropriately localize the noxious stimuli and rank its intensity, but do not experience the stimulus as unpleasant (Grahek 2007 32).[[9]](#footnote-9) The second failing of specificity theory is that there is no evidence for dedicated pain neurons in the central nervous system (CNS), which undermines the postulation of a dedicated pain center in the brain (Moayedi and Davis 213 10).

 The gate control theory of pain, proposed by Melzack and Casey (1968), incorporates the existence of nociceptive receptors, but rejects the one-to-one correspondence between noxious stimulation and the experience of pain. Instead, the gate control theory of pain is the view that distinguishes among different elements of pain, which together form a pain system. The pain system incorporates the sensory and discriminative dimension of pain, which is the intensity, location, quality, and duration of pain; the affective and motivational elements of pain include unpleasantness and the flight response to noxious stimuli; and the cognitive and evaluative aspects of pain, which can mediate the experience of pain based on cultural values, the context in which the stimulus is experienced, and a person’s current state of mind. This multidimensional description of pain is widely accepted in the scientific literature about pain and is the basis of the definition of pain adopted by International Association for the Study of Pain (IASP). [[10]](#footnote-10)

The gate control theory of pain requires the postulation of a gate control mechanism that Melzack and Wall (1965) localize in the spinal cord. This gate control system modulates the input transmitted from nociceptors to the transmission cells (T cells) located in the dorsal horn of the spinal cord. Afferent nerve fibers or receptor neurons carry information from the sensory organs to the central nervous system (CNS), and the dorsal horn is where afferent nerves merge into the CNS. The output of the T cells is based on the intensity of the signal from the afferent nerves. The intensity of the output is the ratio of activation between small and large afferent fibers, the latter of which have an inhibitory effect. The large and small fibers are referred to in most of the literature respectively as C fibers and A-delta fibers (Bishop 1946).[[11]](#footnote-11) The output of the large afferent fibers can also be modulated by the neocortical areas of the brain or by what Melzack and Casey (1968) refer to as the central control system (p. 426). The postulation of the central control system allows for the possibility of the cognitive modulation of pain.

When the output from the dorsal horn T cells is achieved, it is transmitted towards two distinct brain systems: “a) via neospinothalamic fibers into the ventrobasal and posterolateral thalamus and somatosensory cortex; and b) via medially coursing fibers, that comprise a paramedial ascending system, into the reticular formation and medial intralaminar thalamus and the limbic system” (Melzack and Casey 1968 427). These two distinct, but interacting, brain pathways instantiate the three distinct elements of pain mentioned earlier and form the pain system in the brain.[[12]](#footnote-12) The pathway that projects into the thalamus and the somatosensory cortex underlies the sensory and discriminative aspects of pain, while the activation of the reticular formation and the limbic system contributes to the unpleasantness of pain and motivates the person to perform actions required to avoid noxious stimuli. The third element of pain is instantiated in the neocortical areas of the brain and can mediate the experience of pain. Based on Melzack and Casey’s model, the cognitive aspects of pain can have an inhibitory effect and can dampen the output from the dorsal horn, preventing the projection of the pain signal into the brain. But cognition can also inhibit the affective and motivational aspects of the pain system, even after the signal from the T cells has been transmitted through the sensory and affective brain systems. The evidence for the influence of the cognitive inhibitory system is presented in section 3 and assessed in section 4 of this paper.

The distinct elements of pain have been confirmed using functional magnetic resonance imaging (fMRI). In particular, imaging studies have confirmed that the sensory-discriminative and affective aspects of pain are subserved by different brain pathways and that they can dissociate (Rainville 2002).[[13]](#footnote-13) The categories of the affective influence on the experience of pain can be distinguished as pain unpleasantness and suffering, or by what Price calls the “secondary pain effect,” which requires the psychological contextualization of pain in terms of its long-term consequences (Price 2000).[[14]](#footnote-14) According to Price, the intensity and unpleasantness of pain are influenced by different factors (Price 2000, p. 1769). A number of experiments, however, show that pain intensity causes pain unpleasantness. In a study utilizing hypnosis, hypnotic suggestion about pain unpleasantness affected only that aspect of the experience of pain and did not impact intensity ratings. Yet hypnotic suggestion targeted to vary intensity affected both judgments of intensity and of unpleasantness. The secondary effects of pain were shown to be modulated by personality traits, such as neuroticism or extrovertism, with neurotics experiencing more suffering as compared with that of extroverts (Harkins et al. 1989).[[15]](#footnote-15) Intensity of pain, however, remained the same across groups, showing that secondary effects of suffering are not based on the sensory and discriminative aspects of pain.

By moving away from the identification of pain with noxious stimulation, the Melzack and Casey gate control theory can account for the difficult pain phenomena described earlier in the section. Patients whose chronic pain has been treated with a cingulotomy, which is the removal of the cingulate gyrus, a brain area part of the affective and motivation pathway of the pain system, experience pain asymbolia. They are still able to report on the sensory-discriminative dimensions of pain, but no longer feel that the pain is unpleasant. Because gate control theory distinguishes between the pathways responsible for the motivational and affective aspects of pain and the sensory discriminative pathway of pain, it can be used to account for cases where those two elements dissociate. Conversely, damage in the somatosensory cortex has been associated with the loss of the discriminative aspects of the pain experience. Patients with this type of damage report feeling an unpleasant stimulus without being able to accurately locate the source of the feeling or to describe any other aspect of the sensation; for example, whether it is a sharp or a dull pain, or whether it is a burning sensation (Ploner, Freund, and Schnitzler 1999).[[16]](#footnote-16)

 Gate control theory requires a characterization of pain as a more complex phenomenon and Melzack and Casey (1968) propose that pain is a three-dimensional phenomenon. They argue that no individual brain pathway should be identified as the pain center in the brain; rather, the entire interacting system of pathways should be taken to be the instantiation of the pain experience:

Pain varies along both sensory-discriminative and motivational-affective dimensions. The magnitude or intensity along these dimensions, moreover, is influenced by cognitive activities, such as evaluation of the seriousness of the injury. If injury or any other noxious input fails to evoke aversive drive, the experience cannot be labeled as pain. Conversely, anxiety and anguish without somatic input is not pain. Pain must be defined in terms of its sensory, motivational, and central control determinants. Pain, we believe, is a function of the interaction of all three determinants, and cannot be ascribed to any one of them (Melzack and Casey 1968 434).

3. Neurocognitive Modulation of Pain

The three-aspect theory of pain groups together attention, expectation, and learning as potential cognitive influences on the felt experience of pain. In this section, I will present some of the ways in which all those cognitive processes are said to influence felt pain. In section 4, I will evaluate whether the evidence for cognitive modulation establishes the cognitive penetration of the sensory and discriminative aspects of pain.

I will begin by describing the evidence that attention modulates the felt intensity of pain. When an individual is distracted away from a painful experience, the activation in sensory, affective, and cognitive areas of the pain system is decreased (Wiech, Ploner, and Tracey 208 307). A number of functional brain imaging studies demonstrate that distraction away from noxious stimuli can reduce activation in the pain system in the brain areas that subserve the sensory, affective, and cognitive features of pain (Wiech, Ploner, and Tracey 208 307). In a study by Tracey et al. (2002), participants received the same intensity stimulus, but the intensity ratings of the stimulus were lower for the participants who were distracted.[[17]](#footnote-17) Additionally, brain imaging confirmed that the distracted participants had increased inhibitory activity in the midbrain and this inhibitory activity correlated with reports of lower intensity of pain. As distraction can diminish pain intensity, there is evidence that attending to pain can increase the intensity of pain. Chronic pain syndromes, for example, might be the result of hypervigilance for pain.[[18]](#footnote-18)

Expectation has been shown to change how pain is experienced. For example, the application of a low intensity nociceptive stimulus coupled with a warning to expect a high intensity stimulus produces the experience of high intensity pain. Similarly, an expectation of a low intensity stimulus paired with the application of a high intensity stimulus results in a less painful experience (Wiech, Ploner and Tracey 2008). That expectation can modulate the intensity of pain is thought to be at the root of both placebo and nocebo effects. An individual, who has taken an analgesic and has experienced pain relief, will form an association between taking a pill of a certain type and pain relief, and this association will produce pain relief even when an individual is treated with a substance that does not contain any known analgesics.[[19]](#footnote-19) That expectation of pain relief, based on established associations, is the bases of placebo effects is supported by studies that indicate diminished placebo effects in individuals who have been unsuccessfully treated with analgesics in the past.[[20]](#footnote-20)

Expectation can also influence the effectiveness of proven analgesics. In a study investigating the influence of treatment expectation on drug efficacy, Bingel et al. (2011) demonstrated that patient expectations could influence the effectiveness of a pain reliever remifentanil.[[21]](#footnote-21) Positive treatment expectations, i.e., anticipation of pain relief, doubled the analgesic effects of the drug, while negative treatment expectations, i.e., expectation of exacerbation of pain, diminished the pain relieving effects. Bingel et al. (2011) use fMRI to show the nociceptive stimulation resulted in the activation of the pain system. Imaging data was also used to corroborate the reports of pain intensity and to show the effects of analgesia in the brain. Furthermore, Kessner et al. (2013) demonstrated that prior experience with treatment can impact the effectiveness of their current treatment, indicating both the impact of learning and the corollary expectations on pain.

 Another way in which pain can be cognitively modulated is through the reappraisal of the noxious stimulus. The perceived threat of the painful stimulus can affect the intensity of pain and the individual’s assessment of his or her abilities to cope with the threatening stimulus. For example, pain intensity can be affected by the degree of control experienced by the individual. In situations where individuals think that they have greater control, pain is felt as less intense even if the nociceptive input remains the same (Wiech, Ploner, and Tracey 2008). In addition, noxious stimuli perceived to be more life threatening, for example, are ranked as more unpleasant.[[22]](#footnote-22) For individuals with chronic pain, their cognitive appraisal of the pain, whether they felt that their chronic pain negatively affected their life, affected both their coping strategies and their pain intensity.[[23]](#footnote-23) There is also indication that one’s ability to cope through reappraisal depends on personality traits such as neuroticism or extrovertism.[[24]](#footnote-24)

A proposed mechanism for the cognitive modulation of pain is the descending pain modulatory system. The brain regions involved in this modulatory system include the frontal lobe, anterior cingulate cortex, insula, amygdala, hypothalamus, periaqueductal gray (PAG), nucleus cuneiformis (NCF), and rostral ventromedial medulla (Tracey and Mantyh 2007 380).[[25]](#footnote-25) Recent studies confirmed the involvement of the midbrain in the modulation of pain intensity caused by changes in attention. Tracey et al. (2002) showed that changes in pain rating intensity correlated with changes in activity in the PAG, an area in the midbrain. Additionally, the PAG has been shown to inhibit the processing of nociceptive stimuli in the dorsal horn, located in the spinal cord (Eippert F. et al. 2009).[[26]](#footnote-26) The descending pathway of pain modulation underlies both placebo and opioid analgesia.

A study by Eippert et al. (2009) also documented the impact of expectation on processing of nociceptive stimuli in the dorsal horn. Eippert et al. (2009) utilize functional magnetic resonance imaging (fMRI) to show that the application of a painful stimulation could be relived through the administration of a placebo. Moreover, they demonstrated that administration of a placebo correlated with a decrease in response in the dorsal horn (Eippert F. et al. 2009). In effect, the expectation of pain relief resulted in the dampening of nociceptive processing in the spinal cord. The administration of the placebo also resulted in lowered pain ratings. Eippert et al. (2009) hypothesize that their study confirms gate control theory and that the changes in nociceptive processing are possibly due to the activation of the descending pain modulatory system (p.404). As described in section 2 of this paper, the dorsal horn is part of the sensory and discriminative pathway of pain because information about pain intensity is first processed in the spinal cord. Thus, changes in processing in the spinal cord are changes in the sensory and discriminative aspects of pain. Moreover, when those changes are caused by variations in attention or by expectation, they are instances of cognitive modulation.

Both attention and expectation, also, correlate with decrease in activation in other parts of the sensory and discriminative pathway, such as the primary and secondary somatosensory cortices (SI and SII) (Wiech, Ploner, and Tracey 2008). For example, Petrovic et al. (2000) conducted a study to determine whether coupling a cognitively demanding task with nociceptive stimulation would affect the processing of the stimulus throughout the sensory and discriminative regions of the pain system. As part of their experimental design, they asked participants to complete a computerized maze test while administering a standard cold pressor test, i.e., immersion of a hand in freezing water.[[27]](#footnote-27) Utilizing, positron emission tomography (PET), Petrovic et al. (2000) showed that in addition to changes in the PAG, shifting attention to the cognitive task reduced the activation in the somatosensory cortices SI and SII.

Additional brain imaging shows how neocortical processes can recruit the descending pathway of pain to promote analgesia. For example, reappraisal of pain that resulted in lower intensity of pain correlated with increased activity in the right anterior ventromedial prefrontal cortex (VLPFC) (Wiech, Ploner, and Tracey 2008). Similarly, placebo analgesia can increase activity in the prefrontal cortex and decrease activity in parts of the pain system, including thalamus, insula, and ACC (Tracey and Mantyh 2007 382).

Taken together, these studies identifying the regions of the brain that comprise the descending pain modulatory system and they confirm the neurocognitive inhibitory system posited by Melzack and Casey (1968) as part of gate control theory. The evidence supports the view that attention, expectation and reappraisal can influence felt intensity of pain in a variety of different ways. Cognition can inhibit the processing of nociceptive stimuli in the spinal cord and in the midbrain, but it can also affect the processing of sensory and discriminative aspects of pain in the somatosensory cortex. This indicates that cognition can change the perceptual processing of pain all along the sensory and discriminative brain pathway.

An alternative account of the neurocognitive modulation of pain, does not require alterations in sensory processing and is proposed by Wiech et al. (2014). Weich et al. (2014) utilized a probabilistic cuing paradigm to show that changes in pain intensity do not always involve shifts in perceptual processing. In their study, participants were presented with a visual cue that signaled a particular probability--say 80% for a high intensity stimulus and 20% for a low intensity stimulus--that a high or low intensity stimulus will be applied. The participants were asked to identify whether a stimulus is high or low intensity as quickly as possible. Correctly cuing for a high intensity stimulus, for example, resulted in increased accuracy and shorter response time. The error rate response times, i.e., inaccurately identifying a high or low intensity stimulus, was used to determine whether the influence of the cue, which created the expectation of either a high or low stimulus, was due to a change in sensory processing or due to a change in perceptual decision-making. If the cue changed sensory processing the error response times are expected to be shorter than error response times that were due to changes in perceptual decision making. Wiech et al. (2014) noted that the errors in their study resulted in longer response times indicating that cuing resulted in altered perceptual decision-making. They conclude: “incoming sensory information is not analyzed *de novo* but interpreted based on prior information. As a consequence, the incoming information is more likely to be interpreted in accordance with the more likely percept” (Wiech et al. 2014 R680-1).

4. Does Cognitive Modulation Amount to Cognitive Penetration?

In the previous sections, I described a number of studies that substantiate the claim that cognitive processes, such as attention, learning, and memory, modulate felt pain. Based on gate theory, that modulation is exerted through the neocortical influence on the processing of sensory and discriminative aspects of pain. Therefore, the view that cognition modulates the sensory and discriminative aspects of pain is widely accepted in the scientific literature on pain.[[28]](#footnote-28) However, in a recent article, Firestone and Scholl (2016) challenge the view that cognition exerts influence on visual perception. They do not address pain nor do they explicitly liken pain to visual perception. Nonetheless, there are reasons to analogize pain and perception as many in the pain literature characterize pain as a perceptual phenomenon.[[29]](#footnote-29) In this section, I defend the view that cognition penetrates the sensory and discriminative aspects of pain. To accomplish that, I use aspects of the argument presented by Firestone and Scholl to show that the claim for cognitive penetration of pain succeeds even when one takes into account some the common pitfalls they have identified in the literature on visual perception. My argument (and my conclusion) is only about pain. I am not aiming to make any claims about the cognitive penetration of visual processing and my goal is not to mount an argument directly against views espoused by Firestone and Scholl (2016).

 I will focus on three of the purported failings in the literature on perception identified by Firestone and Scholl. One of them is that many instances of cognitive penetration in perception do not result in changed visual processing, which they claim should be required for cognitive penetration. Another claim is that most instances of cognitive penetration in vision are due to peripheral changes in attention, rather than changes in sensory processing. A final claim I plan to evaluate in relation to pain phenomena is that most contemporary instances of cognitive penetration of perception are cases of changed judgment rather than alteration in perception.

I begin by tackling the requirement that cognitive penetration of perception must result in changed perceptual processing and apply it to the case of pain. Taking into account the requirement by Firestone and Scholl, in order for pain to be cognitively penetrated, there should be evidence that cognition changed the processing of the sensory and discriminative aspects of pain. Based on the evidence presented in section 3, there is support for the view that attention and expectation can affect how nociceptive stimuli are processed all along the sensory and discriminative pain pathway. Distraction impacts the processing of pain in the somatosensory cortices (Petrovic et al. 2000). Moreover, distracted individuals have increased activation in the PAG, located in the midbrain, which is known to inhibit the processing of nociceptive stimuli (Tracey et al. 2002). Finally, placebo analgesia inhibits even the early processing of noxious stimuli in the spinal cord (Eippert et al. 2009).

Firestone and Scholl note in their article that evidence of a descending pathway is not evidence for cognitive penetration (Firestone and Scholl 2016 4). What I presented, however, is not merely evidence of a descending pathway for pain, which only establishes the possibility of cognitive penetration. Rather, I cited evidence showing that this descending pathway functions in the way gate theory predicted--which is that neocortical processes can affect the perceptual processing of pain. This then should qualify as cognitive penetration even based on the Firestone and Scholl requirement that instances of cognitive penetration ought to cause changes in perceptual processing.

Firestone and Scholl could dismiss my argument that attention changes perception by stating that it succumbs to one of their stated “pitfalls” in the literature on vision, which is that most cognitive effects in perception are peripheral changes in attention.[[30]](#footnote-30) They argue that it might seem that reasons or motives to attend to a particular stimulus influence perception, but that impression is misleading. They argue that shifting attention away from a stimulus changes experience in the same way as closing one’s eyes would change ones experience, i.e., by changing the input. The actual processing of stimuli remains inflexible, however, and is independent of one’s reasons to attend in a particular way (Firestone and Scholl 2016 13).

Let me evaluate whether this claim applies to the relationship between pain and attention. A number of studies demonstrate that distraction can diminish the intensity of pain.[[31]](#footnote-31) For example, a study by Tracey et al. (2002) that I presented indicated that distraction can impact the early processing of the sensory and discriminative features of nociceptive stimulus. In the study, the participants exposed to a nociceptive stimulus were asked to either attend fully to the stimulus or to “think of something else.” Both groups received the same intensity stimulus, but the intensity ratings of the stimulus were lower for the participants who were distracted from their pain. Moreover, brain imaging in the study confirmed that the distracted participants had increased inhibitory activity in the PAG, which indicated altered processing of the stimulus. Thus, the changes in pain intensity were not the result of changed input because the nociceptive simulation was held constant throughout the experiment.

One could object that even when nociceptive stimulation is held constant, it is possible for an individual to focus on a different aspect of the stimulus, which would result in changed experience without this being the result of cognitive modulation. The shift in attention would change the input, but would not cause changes processing.[[32]](#footnote-32) For example, pain has a number of distinct sensory and discriminative features, such as pain intensity, location, quality, and duration. Changing one’s focus away from pain intensity and instead attending to the quality of the pain, whether it is sharp, dull, or pulsating, might affect how pain is experienced. Almost universally, however, studies on cognitive modulation of pain control for this type of shift in attention by asking participants to report only on one of the sensory features of their pain, i.e., pain intensity. In the study by Wiech et al. (2014), before partaking in the experiment where participants had to make judgments about pain intensity, they were trained to discriminate accurately between nociceptive stimuli based on just that one dimension, which is whether the pain was of high or low intensity. Finally, much of the evidence that shifts in attention affects the perception of pain are coupled with imaging studies that indicate that distraction away from nociceptive stimuli result in decreased activity throughout the pain system, indicating changed processing.[[33]](#footnote-33)

Another pitfall in the visual perception literature identified by Firestone and Scholl is that many of the purported cases of cognitive penetration are likely instances of changes in judgment rather than actually changes in what is perceived (Firestone and Scholl 2016 9). It is important to begin by describing how Firestone and Scholl characterize perception. They state that visual processing is the mental activity that creates *percepts,* which are conscious experiences of the color, shape and size of objects, and perception is the combination of unconscious visual processing and the experience of percepts. They then distinguish between perception and judgment in the following way: “…whereas we can perceive the color or size of some object--say, a shoe--we can only infer or judge that the object is expensive, comfortable, or fashionable…” (Firestone and Scholl 2016 9).

Utilizing the Firestone and Scholl criteria, in order for something to count as cognitive penetration in visual processing, it has to change the percept. Applying this to pain, in order for a cognitive process to penetrate pain, it has to change some of its sensory and discriminative features--say, pain intensity. Even when adhering to this requirement, there is enough evidence to support cognitive penetration of pain. The suppression of pain processing certainly affects the experience of pain intensity, in a way that does not rely on judgment. In fact, the evidence of inhibitory activity in the PAG and in the dorsal horn shows that the changes in experienced intensity after the administration of a placebo, for example, changes nociceptive processing even before it reaches any areas of the brain that could allow an individual to make judgments about painful experiences.

Given that I have invoked changes in the descending pathway of pain processing, I would now like to evaluate whether the alternative interpretation proposed by Wiech et al. (2014) affects the argument for cognitive penetration. Wiech et al. (2014) provide evidence for the view that instead of changes in processing, expectation biases perceptual decision-making. It is not clear whether the authors intend their interpretation to supplant the more widely endorsed claim that cognition changes sensory processing of pain or whether they merely mean to argue that biased perceptual decision-making is an additional way in which cognition can influence felt pain. Either way, I will evaluate this claim in relation to the position put forth by Firestone and Scholl that instances of cognitive penetration are most likely instances of changes in judgment, rather than changes in sensory processing.

The study by Wiech et al. (2014) provides evidence of an inferential process in which information about the likelihood that an upcoming stimulus will be high or low intensity is used to *interpret* the sensory information of a noxious stimulus. Although this view could be used to challenge the argument that cognition alters sensory processing, the claim that sensory and discriminative aspects of pain are experienced through an inferential process does not favor the modularity of perceptual processing built into the view by Firestone and Scholl. Thus, the study by Wiech at al. (2014) is not a repudiation of the cognitive penetration of pain; rather it can be used to challenge the requirement of modularity of perceptual processing fundamental to the view expressed in Firestone and Scholl.

To substantiate this claim, I wish to show how the evidence of biased perceptual decision-making undermines the distinction between judgment and perception. The type of inferential process that underlies perceptual decision-making as Wiech et al. described is not a judgment *about* what is perceived. Here the actual *percept* of pain, i.e., its sensory and discriminative features, is the result of an inferential process. Moreover, this inferential process, although not itself consciously experienced is caused by the conscious experience of a cue that creates the expectation of either a high or low intensity stimulus. This avoids another criticism put forth by Firestone and Scholl against labeling unconscious sensory processes as inferences. They argue against labeling unconscious visual processes as inferences when those processes are based solely on input. In other words, they contest the claim that an automatic, or as they say reflexive, process should be labeled as an inference given that conscious inferences or attitudes do not have any effect on the processing of the input. The study by Wiech et al., however, is not proof of a conscious inferential process--this is why the perception of a high or low intensity stimulus is not a judgment about what is perceived--it is evidence for unconscious inference. The unconscious processing of the stimulus can be accurately characterized as an inference because it is caused by the consciously experienced cue, which creates the expectation and sets up the inferential process. Expectation affects how much information about the stimulus is processed before a decision is reached about its intensity. This is evidenced by quicker and more accurate response times when the stimulus corresponds to the cue and longer response error reaction times. Thus, even when changes in the sensory and discriminative aspects of pain involve biased perceptual decision making, those changes still qualify as instances of cognitive penetration.

But further implications can be drawn from the Wiech et al. (2014) study. One of them is that the distinction between perception and judgment, which Firestone and Scholl describe as intuitive, is not as easy to draw. If the unconscious processing of a percept, or a nociceptive stimulus, can be properly characterized as perceptual decision-making, then judgments are built into to the processing of the sensory and discriminative aspects of pain. This undermines the characterization of nociceptive stimulation as an isolated or modular process. Firestone and Scholl argue that lack of convincing evidence for the cognitive penetration of visual perception is proof of the modularity of early visual processing. Based on the evidence presented in this paper, I conclude that this is not true of the processing of nociceptive stimuli.

5. Conclusion

In this paper, I argue that certain cognitive processes penetrate the sensory and discriminative aspects of pain. I base my argument on the gate theory of pain, which establishes that the phenomenon of pain has three features. Those features include the sensory and discriminative aspects of pain, the affective and motivational aspects of pain, and the cognitive aspects of pain. I provide evidence both for the gate theory of pain and for the claim that cognition affects the sensory and discriminative features of pain. I argue that there is support for the view that a number of cognitive processes, in particular expectation, attention, and learning can modulate the felt experience of pain.

 I argue further that cognition penetrates pain even based on the parameters set by Firestone and Scholl (2016). I describe functional brain imaging data that substantiates the claim that attention and expectation changes perceptual processing of nociceptive stimulation through the descending pain modulatory system. I also consider the view that cognitive modulation is a type of perceptual decision-making. I argue that this alternative account of cognitive modulation of pain is also an instance of cognitive penetration. I accomplish this by showing that biased perceptual decision-making challenges both the distinction between perception and judgment and the modularity of early processing of pain.

1. Wiech, K, Ploner, M., & Tracey, I. (2008). Neurocognitive aspects of pain perception. *Trends in Cognitive Science*, 12(8): 306-313. [↑](#footnote-ref-1)
2. Firestone, C., & Scholl, B. (2016). Cognition does not affect perception: Evaluating the evidence for “top-down” effects. *Behavioral and Brain Sciences,* *39*. doi:10.1017/S0140525X15000965 [↑](#footnote-ref-2)
3. Parts of this section are based on a more extensive description of the pain system in Gligorov, N. (2016). Objectifying Pain. In *Neuroethics and the Scientific Revision of Common Sense*. Springer. [↑](#footnote-ref-3)
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