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DISCLAIMING ANTI-REPRESENTATIONALISM ABOUT PAIN

VERONICA CAMPOS
Federal University of Rio Grande do Sul (UFRGS), BRAZIL
182vkai@gmail.com
https://orcid.org/0000-0001-6242-5600

DANIEL DE LUCA-NORONHA
Jesuit School of Philosophy and Theology (FAJE), BRAZIL
deluca.11@gmail.com
https://orcid.org/0000-0002-4882-5023

Abstract. According to the so-called representational theories of pain, pains are mental states that represent some bodily disturbance. A common argument presented against such theories is that, because many cases of pain are idiopathic (i.e., they are unaccompanied by any seeming disturbance in one's body), they don't represent anything, and hence representationalism about pain cannot be true. Our goal in this paper is to rebuke this argument by showing that evidence from the neurosciences supports the case for representationalism better than they support the case against it.

Keywords: representational theories of pain • nociception • pain • mental states

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1. Preliminary remarks

One popular view about the nature of pain is representationalism. Varieties of representational accounts have been stood up for by people such as Armstrong (1962), Cutter & Tye (2011), and Bain (2017). According to representational theories of mental content, all mental episodes are representations. A branch of those theories that aims to account for pain submits that pains are mental episodes; therefore, they represent worldly states of affairs, not too much unlike sense perception, beliefs, desires, and any other mental state, within a representational framework. What is specific about pains, in these accounts, is that they represent a certain type of disturbance going on in certain parts of one's body. When I, say, have a burning pain in the tip of my left index finger, the pain itself is a mental state that represents the tip of my index finger being burnt. Despite some differences, the aforementioned authors defend what has come to be known as strong representationalism. Basically, this position involves two central theses: (i) mental states of pain are representational because they causally co-vary with specific bodily disturbances; (ii) this causal

co-variation, in turn, is explained in adaptive terms (Coninx 2021). Pain states represent bodily disturbances because the pain system has been naturally selected for this very function, namely, to protect the organism from harm.

In recent years, strong representational accounts of pain have come under attack. They are often accused of being unsatisfactory, mostly because they don't seem capable of accounting for many of the pains that one might encounter throughout one's life. As Jennifer Corns puts it, representational accounts of pain "deliver the wrong verdict in too many cases" (Corns 2020, p.48). The core of the criticism is that such accounts depend on there being a significant correlation between pain and bodily disturbance — a correlation that, as the argument goes, is profoundly lacking. Many times, one has pain without any obvious bodily disturbance. In those cases, there would be nothing represented by the pain. The only way representational theories could account for such cases is by deeming this pain hallucinatory, or illusory, i.e., false. But because cases of pain in the absence of any bodily disturbance are so numerous and prevalent, we would end up saying that a big part of our pain experiences is false, which seems to be an absurd consequence.

Now, the set of examples normally provided in support of this criticism, that is, the alleged counterexamples to representational theories of pain, comprises two subsets of cases: i) cases whereby no tissue damage is found in the precise location where the pain is reported as being, but rather elsewhere (e.g., cases of referred pain and phantom limb pain); and ii) cases where no specific, identifiable damage can be found through medical investigation, even in locations other than the one where the pain is felt as being. Examples of this second variety include pain after healing (when an injury is followed by increased pain after it has healed; see, e.g., Livingston 2012), pain before damage, and various cases of chronic pain, that originate from something other than tissue damage, or persist beyond such damage. If representationalism was correct, the criticism goes, then those pains would be impossible, because of the lack of correlation between what is felt and the presence of a corresponding bodily disturbance.

The problem, however, is that there is empirical evidence pertaining to all of those cases, evidence indicating that there actually is some meaningful correlation between the pain that is felt and some sort of bodily disturbance that the pain connects with. Therefore, we submit, those cases can be re-described in alternative ways, ways that render them compatible with (rather than counterexamples to) representational accounts. In this respect, it should be emphasized that our aim is not to defend strong representationalism. Rather, in the remainder of this paper, our task is to provide descriptions that might render representationalism in a weak sense plausible. To facilitate exposition, we refer to this position as *weak representationalism*.

By "weak representationalism," we mean a view that retains the claim that pains possess representational content, while rejecting the stronger requirement that this

content must exhibit fine-grained accuracy in specifying the exact location, quality, or causal etiology of a disturbance. On this view, the core content of pain is coarse-grained: it represents that some bodily condition has crossed a threshold of potential or actual threat to the organism's integrity. The representational content is determined by the state's role within the pain processing network (its function in signalling deviations from homeostasis and guiding protective action), rather than by one-to-one covariation with specific kinds of tissue damage. Thus, weak representationalism does not require precise spatial or qualitative covariance; instead, it requires only that pain reliably tracks *some* bodily deviation of functional significance, whether peripheral or central, nociceptive or modulatory, actual or impending.

On this account, the only form of covariance that weak representationalism demands is minimal causal sensitivity to states that disrupt or threaten the normal functioning of the organism. These include actual tissue damage, disturbances likely to produce damage, or malfunctions in central nociceptive modulation that impair the system's predictive and protective capacities. Although pain may misrepresent the precise location or sensory quality associated with these disturbances, or its precise severity, it nonetheless retains a reliably triggered relation to conditions that bear biological significance for the organism. The view thereby preserves the intuitive idea that pain is about the body while allowing for empirically well-documented distortions in how that bodily state is experienced.

To ease discussion, we will address two subset of cases separately. Cases in the first subset are the ones in which no tissue damage is found in the precise location where the pain is felt as being, but rather elsewhere. As for those cases, the main outline of our argument is that just because no tissue damage was found where we would expect to find it doesn't mean it doesn't exist. Pain might correctly represent the existence of some tissue damage, but misrepresents the location of the damage. Cases in the second subset, in turn, are those in which no damage exists, anywhere. As for those cases, our argument will be that what pain represents is *some bodily disturbance* of other sorts, that pose no imminent threat to tissue integrity. Such disturbances are incorrectly represented as occurring damage. So in these cases, pain might be correctly representing that there is something wrong with one's body, or body part, but misrepresents the type of disturbance – disturbances that fall short of tissue damage are represented as tissue damage.

Importantly, weak representationalism distinguishes between the representational content of a pain state and the phenomenal manner in which that content is presented to the subject. Pain may therefore be *experienced* as located in a particular body part while *representing* a disturbance whose physical origin lies elsewhere. This divergence is not unique to pain: analogous dissociations are found throughout perception, as in the ventriloquist illusion or lightness illusions, where spatial or qualitative aspects of phenomenal presentation misattribute the underlying worldly cause.

Such cases illustrate that misrepresentation of fine-grained features is compatible with the state's retaining representational content. The interoceptive domain, characterized by coarse spatial mapping and convergent afferent pathways, is especially prone to such distortions.

2. No tissue damage in the precise location where the pain is felt as being

The most emblematic example of this subset is primary (idiopathic) headache. Headaches are broadly classified as primary or secondary. Primary headaches are those with no seeming underlying cause, whereas secondary headaches are a symptom of some underlying pathology (Potter et. al. 2019), such as traumatic brain injury or carbon monoxide poisoning. Since secondary headaches are obviously connected to specific and identifiable damage, it is only primary headaches that pose a challenge to representational theories of pain.

First and foremost, we must concede that in many cases of primary headache, it is true that no disruption is found *in* the head. That is not because there isn't any, but rather because the disruption is to be found "outside", i.e., pain has extra-cranial origin, and it is referred to the head through nerve pathways that, until not very long ago, we didn't know anything about. Those pathways have been observed first in mice (Kosaras et. al. 2009), then in humans (Schueler et. al. 2014; Burstein et. al. 2017).

Take, for instance, migraines, which are a variety of primary headaches. For a long while, it was thought that all migraine episodes originated in the brain and that the accompanying pain depended solely on activation of intracranial nociceptors.[1] (Burstein et. al. 2017; Zhao & Levy 2014). However, as a growing body of evidence came to show, migraines, as well as other forms of chronic headaches, correlate with extra-cranial pathophysiology. Zhao & Levy (2014) found that some headaches originate from inflammatory events in the bones surrounding and protecting the brain. Similarly, Burnstein et. al. (2017) found that some headaches originate from inflammation in the extra-cranial nerves themselves or in tissues they innervate, not only in the pericranial bones but also in the pericranial muscles, and fascia surrounding nerves.[2]

Here is what we are getting at: in at least some cases of primary headache where no damage could be found "in" the head (intracranial damage), extracranial damage of certain type (notably, various sorts of tissue inflammation) was found nearby, "out", i.e., in peripheral tissues that are, nonetheless, anatomically connected with the "inner" side. And it doesn't stop there. Other extracranial events that fall short of proper *tissue damage*, but that can activate nociceptors nonetheless, have also been

found to correlate with headaches.

Some other cases that have once been described as headaches seemingly unaccompanied by intracranial disturbances have recently been found to originate from one or more cranial suture lines (Rozen et. al. 2021). Cranial sutures are the fibrous joints that connect the bones of the skull. They exist to prevent bone separation with external force/injury, and to allow skull movement during cranial growth. Those sutures close with age, typically beginning to fuse in a person's mid-twenties, although some of them can remain only partially fused until later on, sometimes until an individual mid 60's to early 70's (Khandare et. al. 2015). What Rozen et. al. have found is that at least in some cases of chronic headache where no obvious intra or extracranial damage was present, the pain resulted from progressive irritation of nociceptors within unfused cranial suture lines, until they reached a threshold point to generate pain. Possible origins of the initial irritation include remote trauma (i.e., trauma that happened years, sometimes decades ago) and then continued friction with skull plate movement over time, along with subsequent traumas, even microtraumas, such as a hard cough, lying on a pillow the wrong way, etc. Since sutures are partially unfused, even seemingly harmless events like these can eventually elicit the headache, because they activate nociceptors. It is the body's way to make one aware of potential damage to an area that has already been upset by a previous event.

In a somewhat similar fashion, Appenzeller et. al. (1975) found that many cases of migraines were directly correlated with abnormalities in the metabolism of serotonin, as well as with an accumulation of vasoactive substances in the brain. Due to a decrease in certain proteins that would otherwise bring those vasoactive substances back to normal levels, the caliber of both intra- and extracranial blood vessels was suddenly altered, an event that would coincide with the onset of the migraine attack. Whereas, strictly speaking, those events (metabolic abnormalities and enlarged or enshrinked blood vessels) do not qualify as of *tissue damage*, they are biochemical events, nonetheless, that strongly correlate with headaches that were once thought to be completely idiopathic. Again, pain is the body's way to fire a warning, for should these abnormalities in the blood vessels continue for an extended period of time, tissue damage would ensue.

In sum, all those studies reinforce the claim that some headaches, even though not correlated with tissue damage anywhere in the brain, are actually correlated with damage, potential or actual, either within, on the surface, or outside of the cranium. It is true that the mechanism underpinning primary headache is not yet fully understood, but, by and large, some correlation exists between primary headache reports and some disturbance or another. So what we actually have is multiple correlates, rather than one single mechanism underpinning all primary headaches.

Do those findings *prove* that no headache is possible without a correlated disturbance? No. Primary headache that is completely idiopathic might still exist. But as

science progressively uncovers the pool of disturbances that correlate with it, the accumulation of evidence suggests that cases of primary headache that are completely idiopathic might actually come down to a really small number. They're the exception, not the rule.

A similar explanation is capable of shedding light on the phenomenon of referred pain, in a way that is compatible with representationalists' claim that pain represents bodily disturbance. Referred pain consists of a sensation of pain that occurs in a body part other than the one where the injury is. Although it is a very nuanced phenomenon, cases of referred pain are commonly documented involving both muscular or skeletal injuries and tissue damage in internal organs (Arendt-Nielsen et. al., 2000). For example, one of the first symptoms of a heart attack may be pain felt in the jaw or teeth. Likewise, kidney damage can manifest as leg pain, and spinal injuries can manifest as pain in the arm or legs.

These cases might seem like counterexamples to representational accounts of pain, at first glance, since it is difficult to understand how pain would represent tissue damage if the damage is not where the pain represents it as being. However, those cases are not at all different from cases of primary headache, discussed before. The main point is that the representation of referred pain, just like the one in primarily headache, is just not accurate — it misrepresents. Specifically, it misrepresents the precise location of the damage. Damage that is actually in body part X is being represented as being in body part Y. It does not make it the case that representationalism is false, because the abnormal event in the nociceptive system (to which the pain refers) is always there.[3]

In terms of our discussion, it turns out that interoceptive information is just not fine enough to enable the subject to locate pain at the precise site of tissue damage. But, once again, representations can be more or less accurate depending on the degree of finesse of their content. A representation of a referred pain informs us that there is something wrong occurring in the body, even if its contents are not fine enough for the subject to know with 100% accuracy all of its details, such as its level of severity, its primary causes, and, of course, its exact location. This suffices to show that cases of referred pain do not make for counterexamples to representationalism.

It is worth noting that these cases can indeed be understood as involving partial misrepresentation, but partial misrepresentation is neither unexpected nor theoretically problematic for weak representationalism. The nociceptive system evolved under pressures that favor rapid threat detection and action-readiness over fine-grained spatial discrimination. Given the convergence of afferent fibers and the limited spatial resolution of interoceptive mapping, systematic distortions in perceived location, intensity, and qualitative character are biologically predictable. They reflect the inherent constraints of the system, not a failure of its core function. What is reliably preserved is the information that *some disturbance calling for protective action is present,*

even when details concerning where or of what kind are inaccurately rendered.

For these reasons, the representational content of pain should not be individuated solely by the phenomenal location or sensory character reported by the subject. Instead, content is fixed by the role of the state within the organism's broader regulatory architecture: its contribution to anticipatory guidance, sensorimotor adjustment, and homeostatic protection. A pain felt in the leg due to a spinal disturbance represents a bodily deviation requiring defensive or compensatory action, even if the system mislocates the disturbance. In this sense, mislocation concerns only the mode of presentation of the represented disturbance, not the existence of representational content *per se*.

3. No identifiable tissue damage, whatsoever

This section deals with case ii), damage that is only potential is represented as being actual, and case iii), damage that is not there anymore is represented as still being there. Cases of pain before injury are those in which the sensation of pain appears to be caused by the patient's expectations, even when no tissue damage ever ensues (Atlas & Wager 2012). Typically, faced with potential or imminent injury, the patient anticipates the pain they are going to feel, by means of what scientists call a "pain-predictive cue" — a sensory cue that signals an increased probability of imminent pain (Seymour & Dolan 2013). Sometimes, the pain-predictive cue itself is experienced subjectively as pain.

It is worth noting that pain before tissue damage has adaptive value, because it warns one against potential threats. Evolution works to optimize the selection of future actions. Pain-predictive cues do one of two things: they either increase the likelihood of actions that might promote avoidance of future damage, or, alternatively, they numb the pain that is actually going to follow from the damage, when (and if) the damage finally occurs (Fields 2018). When this type of cue is present, nociceptive transmission both triggers and receives feedback from a decision process. In response to the pain-predictive cue, nociceptive transmission will be either activated (the decision to avert potential injury by means of protective action, e.g., removing one's body or body part from the place where it is vulnerable) or it will be inhibited (if the decision is to pursue a conflicting goal, e.g., fighting). In the former, pain before damage is felt precisely so that pain after damage is not (because damage will not occur — it will be precluded by protective action). In the latter, pain before damage is felt so that pain after damage is diminished (it will be silenced by the temporary inhibition of nociception). In other words, this pain that is felt before the occurrence of actual damage puts one's body in an optimal state of readiness for the action decided upon,[4].

In appealing to disturbances "in the body," we do not restrict the notion of disturbance to peripheral tissue damage or nociceptor activation. Contemporary neuroscience has shown that central dysregulation, such as maladaptive plasticity, cortical hypersensitivity, impaired descending modulation, or aberrant feedback integration, as well as inflammation, constitute a physiologically meaningful disruption of the bodily systems that maintain homeostasis and modulate nociceptive signaling. These central abnormalities compromise the organism's capacity to predict, regulate, and appropriately respond to potential threats, and thus qualify as genuine disturbances in the organism's biological economy. Such disturbances fall squarely within the scope of what pain is functionally designed to detect.

This broader conception of disturbance does not trivialize representationalism. Weak representationalism does not claim that *any* neural event causing pain thereby becomes the content of pain. Rather, representational content is constrained by the biological function of the nociceptive system: only deviations that impair its protective or regulatory roles qualify as represented disturbances. By tying content to function rather than to arbitrary neural causes, the view avoids collapsing into an unfalsifiable correlate-tracking theory while remaining fully compatible with established neurophysiology.

It is worth noting that, at first glance, it may seem that phenomena such as pain before damage are entirely psychological, i.e., that it does not originate from the nociceptive system. If this were true, then there would be nothing being represented by the pain (since the damage in question is one that never actually ensued). Corns (2020) would be right that this sort of case poses a threat to representationalism. Nevertheless, research conducted in the past few decades has uncovered the neurological basis of the process involved in pain before damage, and it became clear that there is actually a very distinctive type of bodily disturbance involved (Porro et al. 2002; Steigo et al. 2014; Koenen et al. 2018; Fields 2018; Henderson et al. 2020). Anticipation of pain via pain-predictive cues influences the perception of subsequent noxious stimuli by causing chemical disruption in the brainstem activity, whose job (among others) is to modulate pain under different circumstances. The expectation also causes a state of hyperalgesia because it increases the sensitivity of cortical nociceptive networks. As a result, both brainstem and cortical nociceptors' activities become temporarily abnormal, despite the absence of actual damage.

This is a clear example of what brain scientists call a "top-down mechanism" — a mechanism through which cognitive factors interfere with nociception. In this case, the mental state of expectation of pain (which results from interoception and is itself felt as pain) is a representation. It represents abnormal activity in the brainstem and the cortical nociceptors, which are in a hypersensitive state. The hypersensitivity, it is worth noting, is nothing short of a disturbance – a permanent state of nociceptive hypersensitivity can lead to incapacitating chronic pain; so it is a disturbance that

must be corrected. What we have here is exactly what a representationalist would predict we would have: a mental state (pain) representing a bodily disturbance (hypersensitivity in the nociceptive system).

Now, would cases of nociceptive hypersensitivity, which predispose subjects to feel pain in the absence of tissue damage, be cases of pain hallucinations *because* the damage is absent? Not really. In those cases, as we've been arguing, the subject is representing reality, only he or she is not representing it accurately. He, or she, is getting most of it right, insofar as the disturbance is there, but is misrepresenting the state of the damage this disturbance relates to (damage that is only potential is being represented as though it were actual). Just like a map that represents the city of Paris does so inaccurately, if it places, say, a parking lot where the Eiffel tower is, instead of the Eiffel tower — that doesn't mean the city being represented doesn't exist, nor that the map is not real. The city exists, and the map is real. Only the map is not downright accurate.

A similar explanation allows us to understand phantom limb pain. This is persistent, sometimes chronic, pain in the region of an amputated limb (Subedi & Grossberg 2011). Although there is still no consensus on the precise mechanisms involved in this process, there are two well-established theories. One of them takes into account the activation of second-order neurons in the spinal cord (Subedi & Grossberg 2011). The branches of the axons located in the amputated area form atypical connections with second-order neurons located in the spinal cord. The atypical nature of these connections can be explained by the fact that part of these neurons begin to transmit pain information, even if the limb in question is absent. This process ends up causing increased sensitivity to pain in the area.

The second theory, which interests us more here, focuses on the processing of proprioceptive information in the cerebral cortex (Yoshimura et al. 2023). According to this theory, phantom limb pain has a brain origin: it originates from a disruption of the sensorimotor loops in the cerebral cortex (Mccabe 2005). From this perspective, the sensation of pain is a response to the absence of feedback on motor intentions, which continue to be generated by the brain despite the missing limb. In a famous therapeutic study proposed by Ramachandra (1996), the patient observes a virtual limb, with the help of a mirror, as though the image was there to replace the lost limb. The virtual limb moves as the subject moves the stump. This process activates mirror neurons, which fire both when subjects act and when they observe the actions of other people. The process recruits proprioceptive information stored in memory, such as the location of the lost limb. Such activation, in turn, modulates the proprioceptive process, which then blocks phantom limb pain.[5]

Now, where does representationalism stand in regard to phantom limb pain? Basically, amputation creates a problem in the subjects' proprioceptive system, the system that represents information coming from one's own body. The absence of feedback

from motor intentions (that under normal circumstances is generated by a functional limb) prevents representations of what is going on from being updated throughout body movement. Mirror therapy allows these updates to be re-established, and this in turn, eases the pain. Bottom line is: understanding phantom limb pain *depends on* the very idea of representation. Not much unlike the other cases previously discussed, the subject is getting some of it right and some of it wrong. He or she is getting it right that something is off (something is evidently wrong with his nervous system, since the system is signalling pain as though it was coming from a limb that is no longer there). But he or she is getting it wrong that the damage to which the pain refers originates in the very limb. It is not.

Moreover, representing central malfunction has clear biological utility. Central dysregulation often leaves the organism unable to generate appropriate defensive responses to external noxious stimuli or to regulate peripheral nociceptive input correctly. Pain in such cases functions as a high-level signal that the protective system itself is compromised. Chronic pain syndromes such as CRPS and fibromyalgia exemplify this dynamic: the pain draws attention to dysfunctional regions of the sensorimotor and modulatory circuitry whose impairment places the organism at heightened vulnerability. Pain thereby continues to fulfill its representational and protective functions even when the underlying disturbance is central rather than peripheral.

Last, there are cases of chronic pain and pain after healing, to which the same observations apply (since pain after healing is a variety of chronic pain and is commonly described as such, i.e., in terms of pain that continues to be felt in the absence of an injury. See, for instance, Choinière et al. 1991, and Polycarpou et al. 2005). Evidence suggests that rather than a lack of correlation between pain and bodily disturbances, multiple correlates have been found, and all of them pertain to the central nervous system. Through neuroimaging, researchers have found that patients suffering from some form of chronic pain have disruptions of some sort within brain regions involved in normal modulatory influences on pain (Geha et al. 2008; Lutz et. al. 2008, Tracey & Bushnell 2009), as well as disruptions in brain neurochemistry (Grachev et al. 2002).

In other words, even though a lot remains unknown about the causal mechanisms underpinning each condition that is presently typified as chronic pain (rheumatoid arthritis, fibromyalgia, chronic back pain, etc.), it is established that the brain of chronic pain patients is different. The system that would otherwise modulate pain is faulty, in one way or another, in these patients. For instance, rheumatoid arthritis patients have a lower rate of opioid receptor binding (Jones et al. 1994). Fibromyalgia patients, in turn, have abnormal dopamine responses. They do not release dopamine in response to external pain stimuli, as healthy subjects do (Scott et al. 2006; Wood et al. 2007a, 2007b). And so forth. As Tracey & Bushnell (2009) remark, combined, these studies strongly support the idea that the common denominator among chronic

pain cases is disorder, or dysfunction, in brain neurochemistry. The role played by pain, in this case, is the same as in migraines that follow metabolic abnormalities: it warns one against some disruption in the nervous system, the system that processes and responds to information coming from outside, as well as from within the body.

Now, do those findings support a representational account of pain, do they challenge it, or are they ambiguous, for these prospects? To us, it is clear that they support representational theories, or at least, that they are entirely consistent with a representational description of pain states as events that represent bodily disturbances. In the case of chronic pain, what pain represents, in each disorder, is the respective neurochemical malfunction that turns out to be happening in one's brain in response to stimuli coming from the relevant area in the body. It is as though the pain was saying "something is off with *this* brain region" (the region whose malfunction is reflected in abnormal neurochemical response to stimuli coming from the body part where the pain is felt as being). To be sure, the pain is not felt as being *in* the brain — it is felt as being in the muscle (in the case of fibromyalgia), in the back (in the case of chronic back pain), in the joints (rheumatoid arthritis), and so forth. But what the pain *represents* is a malfunction in the area of the brain that should behave in a certain way in response to feedback generated in those body parts, but fails to. So the patient is not getting it wrong, and his or her pain is not failing to represent.

Representing endogenous disturbances rather than exogenous tissue damage is not unprecedented among sensory systems. Conditions such as tinnitus or visual snow syndrome involve perceptual states that represent internal disruptions of sensory processing while remaining representational in nature. Pain representations of central disturbances fit this broader pattern of sensory systems reporting on their own malfunction. As with these perceptual examples, the mismatch between the site of the disturbance and the phenomenal character of the experience reflects limitations of the sensory system's mapping capacities rather than an absence of representational function.

4. Final Remarks

In the course of this paper, we have shown that evidence from the neurosciences regarding cases where there seems to be a lack of correlation between felt pain, on the one hand, and tissue damage, on the other hand, supports the case for representationalism about pain, or at least is consistent with it. Cases of pain that used to be thought of as being completely idiopathic have had their neurological basis uncovered by research conducted in the past 2 decades, and with this, it becomes clear that in the majority of those cases, there is a bodily disturbance of some sort going on. In such cases of pain, the bodily disturbance is one of three types. It can be i)

actual tissue damage going on in a different part of the body (different than the one where the pain is felt as originating), as it is with referred pain and with most cases of primary headache. Or it can be ii) potential tissue damage that is about to affect the very body part where the pain is felt as being, as it is with cases of pain before damage and some cases of primary headache. Or it can iii) be an abnormal nervous response to nociceptive feedback or its lack thereof, as it is with phantom limb pain and most cases of chronic pain, including pain after healing.

In all of those cases, the pain represents a state of affairs in one's body that qualifies as a disturbance, only it does not represent it with positive accuracy. In i), the presence of actual tissue damage is correctly represented, except for its precise location, which is misrepresented. In ii), damage that is only potential is represented as being actual. And in iii) damage that is not there anymore is represented as still being there. Though the pain in all those cases involves misrepresentation of some sort, they are not gross misrepresentations. I.e., they are not grossly incorrect, insofar as they get a relevant part of the picture right, and only get some information or another wrong. Therefore, the pain in those cases is neither a hallucination nor an illusion. Finally, our defense of representationalism does not commit us to endorsing strong representationalism. Indeed, we are not defending a specific causal co-variation, present in all cases, between pain mental states and bodily disturbances. Rather, we emphasize that pain states are correlated with heterogeneous physiological factors, whose representation exhibits varying degrees of accuracy.

What our account preserves is not a vacuous notion of representation, but a biologically constrained one: pain tracks states that endanger bodily integrity or compromise the organism's capacity for adaptive engagement with its environment. The view is empirically testable (different kinds of disturbances should produce characteristic patterns of nociceptive and modulatory activation) and accommodates both the accuracy and the systematic distortions that empirical work has revealed. Weak representationalism thus retains the explanatory strengths of representational approaches while avoiding the overcommitments that anti-representationalist arguments rightly target.

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Notes

[1]Nociceptors are sensory receptors that are activated by noxious stimuli, i.e., mechanical, thermal, and chemical stimuli that damage or threaten to damage the body's integrity.

[2]Those findings are consistent with results previously encountered by others, for instance, Blau et al (1981) and Schueler et. al. (2013). Blau et al examined fifty patients during headache attack to seek the exact site of the pain, having found that in approximately 50% of cases, pain aetiology had an independent extracranial component, i.e., the pain was being elicited by events that occurred "outside" of the brain.

[3]A quick word might be useful as to how, exactly, pain is referred. Research on referred pain tends to build on top of the so-called "convergence projection theory" (Jin et al., 2022). The biggest part of all reported pain is processed by second-order neurons located in both the spinal and brainstem structures. Sometimes, those structures receive information from more than one efferent nerve (Murray, 2009). According to the convergence projection theory, there is a convergent processing of information coming from the group of nerve fibres that transmit stimuli of touch, pressure, heat and the information coming from the group of nerve fibres that transmit pain signals. We can understand this convergence by highlighting fact that, despite the difference in the type of information, both nerve fibres carry action potentials to the brainstem. This convergence could be illustrated as crossed telephone lines: there is a gap between the location of the pain on the one hand and the site of the injury on the other, but they meet in the "middle". This is what enables pain related to heart tissue damage, for instance, to be projected (and therefore felt) elsewhere in the body.

[4]In addition to the anticipation of pain (that is itself felt as pain) expectations about the size and extent of the damage that could ensue also modulate the intensity with which the stimulus is received. Research carried out by Henderson et. al. (2020) showed that subjects that had different expectations about the pain they would feel at a later time reported feeling more or less pain, according to how severe they thought the damage would be, even when stimuli had, strictly speaking, the same intensity. This might be seen as a further adaptive advantage of pain-predictive cues: they modulate intensity, thus contributing to optimize energy expenditure.

[5]A randomized clinical trial of mirror therapy in leg amputation patients showed a significant benefit compared to the control group (Chan et al., 2007), and so did several other studies, such as a systematic review by Colmenero et. al. (2017) showed.

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